EVOLUTIONARY BIO-DYNAMICS:

From 'the survival of the luckiest' to an evolutionary economics

Klaus Jaffe, 2001

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NOTICE

In the following pages I will try to I present the outlines of a highly complex problem. I apologize in advance, as I am sure that my explanatory abilities are not up to the task of this complex problem. Thus, you may have difficulties in following some (I hope not all) of the arguments presented here. The main reason this will happen is because of the fact that our mind is limited in its ability to follow a dynamic web of a multitude of interdependent variables that act simultaneously on the object of study. In addition, the written narrative obliges us to present ideas in a linear sequence.

Today, with the aid of computers, complex problems can be better analyzed, and relevant insight can be gained, as computers can handle a great number of variables simultaneously (although computers have other limitations). Thus, a symbiosis of computer analysis with human thoughts is the desirable tool for analyzing highly complex systems. The ideas presented here have been developed with the help of the computer program Biodynamica that can be downloaded free at the site http://atta.labb.usb.ve/klaus/klaus.htm

The word Biodynamics has had various meanings assigned to it. Here I am using it in the sense as the term Thermodynamics is used. That is, the science which studies the ever changing interactions among the living.

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INTRODUCTION

Many brilliant new ideas in evolutionary biology have emerged in recent times after the Neo-Darwinian synthesis. For example, the working of the genes has been described as selfish, and plenty of features have acquired a new light with this new description (see for example Dawkin's *Selfish Gene*). The simultaneous evolution of diverse organisms has been compared to the Red Queen from Lewis Carroll's story *Through the Looking Glass,* by viewing at biological organisms as ever evolving (running) entities that stay in the same position relative to that of other organisms, opening the mind to beautiful new explanations for the existence of sex as a means to outwit parasites. And the evolutionary emergence of altruistic behavior has been explained as a natural outcome of evolutionary forces favoring relatives, presenting challenging new theories. Yet, as beautiful and original these ideas are, they are not necessarily true. In order for them to be true, we have to demonstrate that they can be built into scientific theories that are capable of making predictions of mind braking results from plausible experiments. At the end, it is the experiment and the observation of nature, which have to confirm the hypotheses. If we want to follow the scientific method rigorously, we also have to demonstrate that these new ideas, when inserted into biological evolutionary theory, are consistent with all that is known, and that they can be imbedded into accepted science without violating general mathematical and physical principles. It is these scientific tests that are not necessarily met by these beautiful new ideas. But in the process of testing the rigorous scientific criteria for new theories, these ideas have shown they heuristic value as they produced new and deeper insight into the working of nature and specifically of biological evolution. Here I want to present a synthetic overview of one such exercise resulting in new insights into evolutionary theory.

Publishing in peer-reviewed journals, when trying to present new interdisciplinary ideas, is a very painful exercise. After months and even years of discussions with referees, a paper is finally accepted. The accepted version of the paper often resembles little to what the author originally wanted to present. Although referees undoubtedly add value to the papers, in the cases of new complex and controversial ideas, these have normally to be dropped. This procedure is helpful in simplifying the life of science readers, presenting them with uncontroversial written evidences of new discoveries. But this procedure does not help innovation and creativity. After publishing over a centenary of papers, I am left with the feeling that my contribution to theoretical evolutionary theory is diluted and unintelligibly distributed in mutilated manuscripts in dispersed journals worldwide. Thus, I thought it useful for students of evolutionary biology to have a possibility to read a more coherent synthesis of what I call Biodynamics. As our electronic age allows to publish material which otherwise, due to economic and commercial considerations, would be un-publishable, here is the material, which can be used *at libitum* and distributed free with the only petition as to mention its origin.

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1 The dynamics of biological evolution: an overview

Biological evolutionary theory, already envisaged by Leonardo da Vinci (1452-1519) in a rudimentary form, by giving a correct meaning and interpretation to fossil organisms, was founded on the pioneering works, among others, of Jean Babtiste Lamark (1744-1829), who was the first to envisage evolution as a process guided by innumerable small steps of change; of Charles Lyell (1797-1875), who recognized various geological eras; of Herbert Spencer (1820-1903), who recognized the power of selection in the dynamics of change; of Alfred Russel Wallace (1823-1913) and Charles Darwin (1809-1882), who formulated a theory for the origin of species; and of Gregor Mendel (1822-1884), who discovered the quantum nature of inherited information. Thanks to new insights gained by later discoveries in genetics, molecular biology and population ecology, these first concepts of evolutionary theory were integrated into a more rigorous conceptual theory by, among many others, Theodosius Dobzhansky (1900-1975), Enst Mayr and Douglas Futuyama, forming what is today recognized as the Neodarwinian synthesis of biological evolution. This powerful theory has benefited from mathematical conceptualization promoted by the works of Ronald A. Fisher (1890-1962), M. Kimura, John M. Maynard Smith, William D. Hamilton and many others. Out of these efforts, a consensus emerged that in general terms, biological evolution is based on three fundamental processes: Vertical transmission of genetic information, random mutations in the genetic code and natural selection. Each of these processes are studied nowadays by different scientific disciplines. Genetics for example has uncovered some complex processes occurring in the transmission of genetic information among organisms of species with different reproductive systems. Molecular biology has improved our understanding of how the genetic information is stored in macromolecules and has unveiled biotic and abiotic mechanisms that produce mutations. Regarding natural selection, the study of the dynamics of populations and that of the ecological webs surrounding life have allowed for numerous refinements of our concepts on biological fitness.

However, every one of the three basic processes defining biological evolution has been questioned as to its generality and absolute validity. For example, the discovery of horizontal transmission of genetic information in Eubacteria and Archaria and the existence of cytoplasmic information that is transmitted to offspring in Eucaria, force a revision of the conceptual foundations of evolutionary theory. Refining evolutionary theory so as to account for occasional horizontal transmission of genetic information, selective modulation of mutations and sexual selection will certainly improve the predictive powers of the theory.

Nevertheless, biological evolutionary theory, for the moment at least, is the only general theory available to biologists. Despite its long success as an heuristic tool in explaining the large variety and forms of known life, it has failed so far in explaining unambiguously many important facts of life, such as the origin and maintenance of sex, the emergence and maintenance of social behavior, and the existence of altruistic behaviors. A broader and more interdisciplinary approach to the study of biological evolution may improve the theory and thus, may eventually increase its usefulness in advancing our understanding of life. One of the main challenges left to theoretical evolutionary biologists is to complete and/or rebuild evolutionary theory so as to convert it into a quantitative scientific theoretical framework that promotes experimentally testable predictions. Until now, the predictive power of evolutionary theory lacks strongly behind it's *a posteriori* explanatory power. That is, we may explain evolutionary history by using evolutionary theory, but evolutionary theory is grossly inadequate for quantitative and even qualitative predictions of the outcome of evolutionary processes occurring at present. For example, classical evolutionary theory is of very limited use if we want to predict the outcome of human or environmental influences on a specific biota, or if we want to predict the scope and limits of future biological evolution on extant organisms.

Biological evolution is certainly a complex process, as it involves the action and interaction of many different components, and in addition, its dynamics has an irreversible nature. From a purely mathematical standpoint of view, an analytical search for a putative optimum combination of genetic traits reached by evolutionary mechanisms acting on a complex assemblage of genes, mimicking real organisms, is not possible at present, and may be impossible to achieve even in principle. In addition, we know that stochastic and chaotic processes influence the final outcome of natural selection. Natural selection not always selects the optimal available solution, but often favors the "luckiest" one, i.e., evolutionary solutions are often given by the history, geography and the dynamic constraints of a particular biological process rather than by the theoretical parsimony and efficiency of the selected alternative. It is chance in addition to adaptation that allows a species to establish itself among the living.

The study of these complex properties of biological evolution has been restrained by the scientific and technical tools we had available. Luckily, recent advances in computer sciences and in non-linear mathematics and thermodynamics, offer us tools that have shown to be valuable in solving similarly complex problems in chemistry, engineering, geology, astronomy and meteorology, for example. Among these tools we may name far-from-equilibrium thermodynamics, artificial intelligence and stochastic agent based computer simulations.

Using a stochastic computer simulation model for the study of the population dynamics of multi-loci agents (virtual organisms possessing multiple genes), the present work attempts to show that, by incorporating concepts from thermodynamics and from complexity theory, a revamped biological evolutionary theory may, not only explain the emergence and maintenance of sex and social behavior, but might eventually become a predictive quantitative mathematical (or computational) scientific theory, and so increase enormously its heuristic value.

The main innovation in our way to look at the changing patterns of life on earth, which is revealed *a posteriori*, after applying this approach to the study of biological evolution, is that natural selection, although very important is not the only force regulating evolution. Other factors, such as history, mechanisms optimizing variability and evolutionary features allowing organisms to capture opportunity, are also important. We know that the stochasticity of the evolutionary process is not based solely on the randomness of mutations but also on a certain randomness of the selection process. Thus, rather that speaking of the survival of the fittest, it would seem more appropriate to refer to the process of biological evolution as the survival of the luckiest. Luck certainly is aided by the fitness or preparedness of an individual to cope with its environment, but it also has important stochastic flavors.

The following two chapters will introduce and define some general concepts, relevant for a more modern understanding of biological evolution. Specifically, Chapter 2 analyses some of the limits of the traditional approach in the study of biological evolution and Chapter 3 will define complexity and introduce some relevant thermodynamic concepts in the study of biological evolution.

In Chapter 4, the model Biodynamica is formally explained. The remaining chapters of the book are written without expecting the reader to understand the details of the simulation models used, but certainly reading Chapter 4, even superficially, may help understanding the following chapters.

In Chapters 5 and 6, I present my solutions to the mysteries of why the sexes are always two (Chapter 5) and why sex exists at all (chapter 6). Then, in chapter 7, I explore with simulation results, the dynamic properties of various different mate selection strategies and their effect on the evolution of sexual organisms. Sex.

Chapter 8 tackles the mystery of the existence of social behaviors by exploring dynamic constraints in the emergence and maintenance of altruistic behavior in the development of parental care. The results obtained from the simulations presented in this chapter allow us to dream of a conceptual bridge, joining the biological sciences with economic sciences, and thus eventually starting bridging the gap between natural and social sciences. This is attempted in Chapter 9 by presenting experimental data

relevant to the thermodynamics of social evolution. In Chapter 10 I speculate on the possibilities of a unifying probabilistic quantitative evolutionary science.

2 The limits of reductionism in the study of biological evolution

ABSTRACT: Simple systems are those that can be described completely in analytical mathematical terms. A reductionist approach to scientific research has favored the consecutive dissection of complex system into simpler ones, until they can be described analytically. Such an approach has helped in the advance of physical, chemical and biological sciences, but has been less useful in our understanding of biological evolution. This section tries to convince the reader that significant advances of our present state of the art regarding our knowledge of biological evolution will not be achieved with more conceptual simplifications. Simplification of biological evolution in order to grasp it analytically, although in principle desirable and convenient from the stand point of the classical mathematical researcher, is not recommended at present, as it has lead to distance theoretical evolutionary biology from biological reality and has lead to predictions which have been falsified by biological experiments. The present section tries to detect some of the more dangerous and conspicuous simplifications commonly made in theoretical evolutionary biology.

SIMPLIFICATIONS IN EVOLUTIONARY THEORY

Science has achieved many important or even impressive successes. Among them we may, without much doubt, count the advances in our understanding of the physical nature of the interaction between simple bodies, as evidenced by the success of classical, statistical, relativistic and quantum physics. This success was aided by the development of analytic mathematical tools that facilitated a thorough description of these processes. In this respect, classical mechanics has probably achieved the most comprehensive mathematical description of its research subject if compared to that of other sciences. This success in mechanical physics and other fields has made it very attractive among scientist to try to apply the vast mathematical knowledge gained that way to more complex systems, including biological ones. In order to do so, complex systems are decomposed into simpler parts until they are amenable to mathematical analysis. This approach has received the name of ***reductionism***. Based on this approach, we might define a ***simple system*** as a system that is amenable to a complete analytical formulation. A simple system is in principle a mathematically completely comprehensible entity. Simple systems are relatively easy to describe and are eventually amenable to an intuitive understanding by our minds. Therefore, reducing reality to a set of simple systems is a very attractive alternative if we want to help opening reality to our understanding.

Following this reductionist vein, ***classical evolutionary theory*** assumes that biological evolution can be achieved through three basic steps or fundamental processes, which are thought to be necessary and sufficient to explain biological adaptation. These fundamental processes are:

- Vertical transmission of genetic information

- Random variation through mutation of genes

- Selection of organisms by the survival of the fittest

The simplified version of biological evolutionary theory assumes that these three processes, working together, achieve a continuous change in the genetic makeup of organisms that allows them to adapt to a practically unlimited variety of environments. In this context, biological evolution is seen as a continuous process that allows the emergence and existence of a practically infinite variety of organisms,

each adapting to specific ecological niches. This version of biological evolution assumes that the final phenotype upon which natural selection acts, is dependent on the underlying genotype, expressed phenotypically following epigenetic rules. As *epigenesis* we understand the process by which DNA molecules (deoxyribonucleic acid polymers) in each cell nuclei, express the information they have accumulated and encrypted during evolution, by synthesizing RNA molecules (ribonucleic acid polymers), which in turn code for the synthesis of protein molecules, which in turn synthesize metabolic products, which build cellular structures, which then serve as the basis for physiological processes, which then produce specific behaviors of the organism on which natural selection will act.

The limits of evolutionary theory

A reductionist approach to the study of evolution consists in dissecting conceptually the component parts of the evolutionary process into simple components, amenable to analytical descriptions. Such simple conceptual components include:

Genomes or the substrate for the storage of genetic information. These consist in reality of a complex assemblage of coded sequences of nucleic acids, forming strings of DNA molecules, which assemble to form the chromosomes. For modeling purposes, a genome is often simplified as a continuous string of four bases whose sequences code the genetic information. Among the most popular of such models are those based on binary codes (Holland 1960), which are used for building genetic algorithms used in models of artificial intelligence (see also Kauffman 1993). In such strings, new information is produced by random mutations of single bases, which are substituted by one of the alternative three bases (i.e. base substitution), or by base deletions, by base insertions or by the cross-over of whole fragments of the genome which are interchanged with fragments from a different genome.

Genes are units of the genome that may code for a specific messenger RNA and often for a specific protein, although they may code for more than one protein or for no protein at all. The genome is segmented into discrete parts that are generally delimited by long runs of coding triplets without stop codons called open reading frames. Several genomes of extant organisms have been completely sequenced and thus we know that we might expect genomes with 400 to 8000 genes for microorganisms and up to 150,000 genes for multicellular organisms such as vertebrates and ourselves. Many of such genes have their origin in the duplication of a functioning gene, which then mutate and evolve independently, eventually coding for new and different functions. This process allows us to identify certain families of genes, which share a large percentage of identical sequences (normally over 40 % of the total sequence), but which not always have the same nor similar function.

Loci are sites in the genome that may suffer mutations. Here, I will make no distinction between loci and genes, although we may imagine a single gene to have various loci, where mutations may have different effects on the expressed phenotype.

Alleles are copies of genes with the same or a similar function but with slightly different codes, so that one or a few characteristics of the function for which the gene codes may differ between different alleles. Organisms have only one allele if haploid, or two alleles if diploid, of each gene.

The reductionist or simplified version of neodarwinian evolutionary theory assumes that the process of genetic evolution produces variations in the frequency of alleles in a given population through three basic processes: random drift, migration and natural selection.

Random genetic drift is probably the main source of genetic variation between populations and species, at least among humans (Cavali-Sforza 1994). Changes due to genetic drift in the allelic frequency at a given loci occur as a consequence of selection changing the allelic frequency at another loci. This occurs if both loci are associated by chance in a given population (individuals in the founder population had the

same alleles in the two loci, for example). Genetic drift is more important the smaller the population. Genetic drift causes variations in the proportion of alleles a given populations maintains, so that these allelic frequencies are not due to natural selection. The existence of genetic drift provides irreversible properties to the evolutionary process (see below).

Migration is the process by which a given population receives genes or alleles from a population that has suffered an independent process of evolution during a given period of time, thanks to individuals from both populations that cross breed.

Natural selection will act in order to select the alleles that confer the organism a greater adaptive ability, and in this process may increase or decrease the frequency of specific alleles, eventually driving them to complete *fixation* or extinction. In some cases, natural selection may favor the maintenance of specific proportions of a set of alleles in the population by favoring the survival of heterozygous individuals. A classical example of the heterozygous advantage is the resistance to malaria conferred by the maintenance of a mix of hemoglobin proteins in human populations.

Reductionist evolutionary theory has probably reached its most powerful expression in *quantitative population genetics*, where analytical mathematical formulations allow for the quantification of variations in allele frequencies in target populations. Analytical formulations, such as the Hardy - Weinberg – Castele equation, have shown to be useful as a first approach in our understanding of population genetics. However, in order to solve these analytical formulations several simplifications are required. These simplifications are not always realistic in view of what occurs in natural populations and may distort our understanding of adaptation in real populations of organisms. Among these simplifications, the most critical may be:

Few genes at a time are studied in these formulations (normally one or two), yet real situations of adaptation may involve a large number of genes.

Random mating is normally assumed in these formulations, but mating is very rarely random in nature (see Chapter 5 and 6).

Smooth stationary fitness landscapes. Most idealizations and models of biological evolution assume that given a specific environment, adaptation will favor a specific and unique combination of traits that will allow optimal survival of the organism in this environment. That is, the optimal fitness coordinates are fixed and unique. Adaptation in such fitness landscapes can lead to the optimal peak by increasing individual fitness in small evolutionary steps. History, though, teaches us that fitness landscapes change over time, and normally have multiple adaptive peaks at any given moment in time, i.e. real fitness landscapes are dynamic and rough.

Constant selection pressure has to be assumed in most analytical formulations of adaptation, although it is clear that selection pressures very often change in time and space, with various, often contradictory pressures acting on the populations, forming rather *rough dynamic fitness landscapes*, often ever changing, with no clear single optimal strategies to which adaptation may lead.

No interaction among genes or very simple interactions between genes have to be assumed in order to obtain analytical mathematical solutions in population genetics. Yet real life often involves complex and non-linear interactions among genes that cannot be studied with linear analytical mathematical formulations.

Other features, which are normally ignored in reductionist analytical evolutionary theory but which are known to be present in living systems, are:

Multiple adaptation that imply that a given information in a gene, selected and fixed through adaptation, may serve multiple purposes, not often evidenced from the study of organisms in their present

environment or the environment in which they are studied.

Sexual selection is clearly at work in most, if not all sexual organisms. Despite the fact that the scope and importance of sexual selection, in relation to other features of natural selection, is not well known nor can be defined unambiguously, it has to be taken into account if we want to advance in our understanding of the evolutionary process. This aspect will be the study of Chapter 6.

Horizontal transmission of genetic information is known to occur. We know that plasmids and viruses (mainly retroviruses) are able to insert pieces of genetic information in the genome, transmitting genetic information between organisms without requiring sexual contact. Its importance has so far been underestimated but it may be much more common than previously thought (Doolittle 1998). Another example of accumulation of genetic information through processes different to vertical genetic inheritance is the putative origin of eukaryotes through the chimaeric merging of a eubacterium and an archaebacterium (Gupta and Golding 1993, Koonin et al. 1997).

Cytoplasmic transmission of information: Information not stored in nucleic acids, but present in the cytoplasm of gametes, may be transmitted to the germinal cell from which it modulates the development and the embryogenesis of the new organism. A curious example of cytoplamatic transmission of information is called sexual imprinting (Iwassa 1998), where alleles are suppressed or expressed depending on if they were inherited from the father or from the mother. More instances of cytoplasmatic transmission of information are probably going to be uncovered in the future.

Pseudo-randomness: No known device or algorithm produces infinite series of truly random numbers. This may be because no truly stochastic processes exist in nature and what we call random processes are just processes for which we have no information, or have insufficient information about the boundary conditions. The fact that we are not able to design truly random series of numbers might be a consequence of our limited minds. If we proceed heuristically as in thermodynamics, our inability to produce truly random series of numbers should be the basis for the formulation of a physical law, which would state that "no truly random processes exist in nature". Whatever the outcome of our search for "true randomness", we need the concept of randomness or stochasticity in order to model reality. As we cannot assure that true randomness exist, the word pseudo-random seems to be more appropriate. We will assume here that some processes are random in the understanding that pseudo-randomness is meant, retaining an agnostic attitude towards the true existence of randomness until a better explanation is available.

Punctuated equilibria. Irregular non-linear evolutionary rates are very likely to occur and may have been the rule in the history of life on earth. Gould and Lewontin (1979) proposed that evolution over geological time occurred in bursts of rapid speciation; most species of a given geological era evolve over short periods in geological time, followed by long periods of stasis where the number and form of extant species change little (Sepkoski 1993). In this view, periods of stasis were disrupted by geological catastrophes, eliminating a large proportion of species, giving rise to new bursts of speciation. This irregular dynamic pattern produced the geological strata that mark geological eras. If this view of biological evolution is correct, then the classical working of adaptation, as studied in microevolution, may not explain the natural history of extant and extinct organisms as observed in macroevolution. If the present species composition of the biota has been the product of macroevolution rather than of microevolution, or if the present biota is the product of both, we have to include macroevolutionary dynamic mechanisms in our models if we want to have a better grasp of the history of life on earth.

SOME CONCEPTS WHICH MAY HELP OVERCOME REDUCTIONISM

Biological evolution and the biota, viewed as a dynamic system, show formidable degrees of complexity. In order to mirror parts of this complexity in theoretical models, the following dynamic properties have been invoked in order to explain some important features of biological evolution:

Positive feedback and population explosions

Many biological processes have positive feedback mechanisms. A classical example is unrestricted growth of populations as formalized by logistic growth equations. That is, if the total production of offspring in the life span of an organisms is greater than that required for the replacement of the extant population, the resulting growth curve will be exponential. This type of kinetics is analogous to exothermal chemical oxidation or to nuclear fusion, which has been successfully described mathematically in other sciences.

Negative feedback and regulation

Most physiological mechanisms have negative feedback controls. That is, processes respond in a way that limits the maximal value of certain variables. This regulation is often achieved by dampening the input of the signal as it increases in intensity, obtaining a more constant output. Examples of negative feedback regulation are, at the ecological level, the drop of foraging activity with increased food intake in many animal species, and at the molecular level, metabolic processes, beautifully described by biochemists, such as the Krebs cycle.

Homeostasis

Regulatory processes exist which involve both negative and positive feedback mechanisms, producing a constant output (behavior) regardless of the input (environmental fluctuations). Examples of homeostatic processes are the body temperature regulation in endothermic animals, where mechanisms which increase heat production are triggered when body temperature drops in cold weather, such as uncontrolled muscle contractions producing shivering, and mechanisms such as sweating are triggered when body temperature rises above certain thresholds.

THE LEVELS OF SELECTION.

Nature selects favorable traits at many hierarchical levels. The first level where natural selection may occur is at the level of genes. Competition between genes and differential survival of genes may be called *gene selection* (Dawkins 1989). Classical natural selection theory, though, assumes that selection acts mainly at the level of the *organism.* Organisms compete between each other, showing differential survival, and are thus able to adapt to changing environments or to new environments. Hamilton (1964) introduced the notion of *kin selection*, which assumes that the population of organisms sharing a given allele or gene, may be considered as an unit of selection, as any of those organisms which manage to survive to reproduce will transmit that gene to future populations. Kin selection can be viewed as an intermediate level of selection between that of the organisms and that of populations. *Population or group selection* has been invoked to explain features in organisms that are not easily understood if we assume that selection acts only at lower levels. Features such as sex, altruism and social life are easily explained by assuming that such characters confer advantages to populations possessing them. For selection to act at the level of populations, we must assume that certain populations are reasonably isolated reproductively, that they compete among each other, and that differential survival among these populations exists. The conditions for the existence of natural selection at the population level may not always be achieved in nature, and thus, group selection has not been the preferred explanatory selection mechanism for most biologists. *Species selection* has been invoked several times in the history of biological sciences. If we assume that the great majority of species that ever existed are extinct (Gould and Lewontin 1979), it is difficult to negate that differential survival of species occurs. Competition between species is a common phenomenon in nature. Thus, selection at the species level cannot be ignored. Species selection is assumed to be the subject of study of macroevolution, but the distinction between macro- and microevolution is rather diffuse. Biological processes occur at various different time scales simultaneously. Therefore, biological evolution can only be understood if the dynamic properties of the different levels of selection are taken into account.

THE DYNAMIC NATURE OF BIOLOGICAL EVOLUTION

Biological evolution is a dynamic process in which time is a fundamental variable. Reductionist evolutionary theory has to assume that processes leading to adaptation are constant in time. An additional difficulty with time in biology is that time is irreversible (see below). In biology, the future is not equivalent to the past, whereas physicists commonly assume symmetric time reversal. This fundamental difference makes it conceptually impossible to translate analytical models directly from physics to biology. In this respect, evolutionary analysis based on classical kinetics is limited due to the irreversible nature of biological evolution. The irreversible nature of biological evolution is not only due to existence of dynamic rough fitness landscapes, but is also based on the fundamental asymmetry in the conception of biological time.

Adaptive processes may be difficult to track due to the simultaneous occurrence of different evolutionary processes. For example some characters possessed by different species may converge to similar functions and form (the eye for example) and we have ***convergent evolution*** producing analogous characteristics. At the same time, other characters may suffer ***divergent evolution***, each process taking place with a different rate. Physical and thermodynamic constraints of biochemical and mechanical processes may favor convergent evolution, whereas chaos, randomness and irreversibility may favor divergent evolution. As biological characters produced by these divergent processes interact in an organism, their study outstretches the limits of reductionism. New heuristic approaches need to be developed for the study of complex systems, such as those involved in biological evolution.

Evolutionary irreversibility and the limits of selection

Not all properties observed in extant organisms are the product of adaptation. Genetic drift, neutral evolution, self-organization of complex assemblages of simple interacting parts, and other processes that emerge with the increase in complexity of the biological systems, constrain and modulate biological evolution. When organisms consist of over 100,000 genes, the process by which species acquire through evolution a certain combination of interacting genes, is not likely to be reversed, and the probability of such a reversal of evolution happening by chance is, in all practical terms, zero. Even known reversals of biological characters, such as the evolutionary return to aquatic habits by some mammals, or the loss of vision by cave animals, are clearly recognized as "irreversible evolutionary reversals", because several characters possessed of these organisms reveal the former way of life, and because these "evolutionary reversals" clearly followed a different path to the evolutionary route that lead organisms out of the water or to acquire vision, in the first place.

Not all the genetic features forming extant organisms are the parsimonious product of optimizing evolution. All living organisms are members of a species, which has been derived from other species, and has its genealogical roots at the beginning of life. These species, or its predecessors, have survived several environmental, geological and astronomical catastrophes for which no biological adaptation is possible (it would be rather far fetched to claim that extant species have adapted to survive massive volcano eruptions or giant asteroid impacts). Therefore, we should expect to find in all extant species, features that have survived rather by luck than through evolutionary adaptation. Given the large number and variety of genes and the complexity of their interactions, it seems reasonable to assume that a great number of the genetic features found in extant organisms, are not so much the product of natural selection. i.e. of the ***survival of the fittest***, but rather the product of a process we may call the ***survival of the luckiest***.

3 On complexity, thermodynamics and biological evolution

ABSTRACT: Biological evolution is a rather complex phenomenon and thus, reductionist analysis of the evolutionary process may not fully explain real life. Different scientific disciplines, specially thermodynamics, informatics and engineering, have developed tools which allow us to penetrate into the amazing web of entangled information imbedded in complex systems. Here I explore some of those tools and analyze them in respect to their amenability or usefulness for the study of biological evolution. But for a want of clear guidelines in the endeavor of mixing different sciences, in order to synthesize new tools for the study of biological evolution, a precise rooting of these new ideas into the general conceptual framework of science is required.

GENERAL PRINCIPLES IN SCIENCE

We have to define certain general principles, in order to provide a philosophical base, on which to build a conceptual framework, with which a better model of biological evolution can be constructed. Here we will assume that these general principles are true *a priori* or of undeniable heuristic value. These assumptions are normally thought of as the philosophical principles at the ***origins of science***.

Realism and rationalism:

The theory that states that there is a world of material things in space, which do not depend for their existence on philosophical idealism, is what I will call realism. In perception we are as a rule directly or non-inferentially aware of material objects that exist independently of us. It does not imply that we always perceive things as they really are, nor does it imply that we ever perceive more than a small selection of what is true about material objects or processes. Rationalism then may be defined as the philosophical attitude, which denies the acceptability of beliefs, founded on anything but experience and reasoning, deductive or inductive (Russel 1960).

Scientific agnosticism and falsifiability

Scientific agnosticism is the philosophical attitude, which avoids speculations, so that on all matters, unless rational science can speak, silence is the only wisdom. Intellectual progress is achieved by the successive interplay between induction (reasoning involving generalizations) and deduction (reasoning deriving the truth from simpler causes). When one such process is not possible because of a lack of information available, a scientific agnostic attitude is to go on with other problems that might be tractable with an inductive – deductive process. Going a step further, we might add to scientific agnosticism the concept of falsifiability. Falsifiability, as defined by Popper (1934), is a character of a theory that allows it to be tested or to be falsified. Theories, which do not have this character, are not scientific, as they do not allow advancing knowledge by disproving or confirming existent theories. I believe that a scientific theory, in addition to have an agnostic character, should be formulated such that it can be shown to be false (preferably through experiments), as this is the only way to advance our knowledge in the direction of the material world and not in the direction of our pre-conceptualized (by brain and culture) minds.

Complex scientific theories can be falsifiable in many different ways, as each component part of the system may be falsifiable. Thus additional criteria are needed in order to select among various competing or alternative complex scientific theories. One such criteria, I propose, is **predictability**. That is, a theory is better than another, if it is able to predict more new phenomena or the outcome of future experiments better than the competitors. This criterion may be especially relevant in evolutionary theory, where most of the great evolutionary experiment lies behind us. Any evolutionary theory should improve our understanding of the phylogenetic relationships of living organisms, and if possible, provide quantitative predictions, which can be tested with actual data. Prediction here does not necessary mean

the provision of a description of a phenomenon that has not yet occurred, but might also refer to descriptions of specific features or relationships of phenomena which have already occurred (like evolution of extant and extinct organisms) but which had not been described before.

What is a complex system

In contrast to a simple system (see above), we may define complex systems as those that are not amenable to a complete analytical description due to limitations of the mathematical tools available, or due to fundamental aspects of their structure which do not allow the formulation of precise analytical relationships. This definition is analogous to the *many body problem* defined in physics. As Henri Poincaré (1854-1922) pointed out, problems of three or more interacting bodies have normally no exact analytical description, placing them into a category outside of the absolute grasp of mathematics. These categories of systems are referred to here as complex.

Why is biological evolution complex?

Biological evolution has many features, which makes it a complex system.

- It is a dynamic process where variables and their interaction change with time and space.
- It is a system where a great number of variables interact. For example, an organism which has 2000 genes with allelic variance (most Eubacteria and Archaria have many more), with an average of 10 different alleles per loci (gene) has to test 10^{2000} possible combinations of alleles in order to find the optimum or best solution for a given environment with a specific fitness landscape. This number exceeds by far the total number of electrons in our universe.
- Adaptation occurs in rough variable fitness landscapes, changing in time and space. Given the nature of genetic information, several optimal combinations of genes may be feasible for a given point in the fitness landscape. Such systems are typically complex and are analogous to the spin glass models, to the dynamics of turbulent fluids, to the physics of climatic change, and many other complex physical problems (Ruelle 1975).

Tools for the study of complex systems

Real systems are almost always complex. Thus, tackling real complex systems like chemical multi-compound reactions, hydrodynamics (specially turbulence phenomena), "spinning glasses" or changes in viscous dynamic systems, climate predictions, soil dynamics, geological history, economy, etc. have forced engineers and to a lesser degree physical and chemical scientist, to design tools for tackling these processes. Among the tools these disciplines developed to study complex systems are:

Chaos, attractors, fractals, singularities, catastrophe theory and other paraphernalia.

Mathematical sciences have been able to describe analytically some phenomena, which share some characteristics with complex systems, or which have some resemblance to complex systems. Among these descriptors are:

- **Chaos** refers to the apparent random changes suffered by some dynamic systems. Chaotic dynamics is not random however, but is in principle predictable, although it is very susceptible to border or initial conditions. Chaotic phenomena can, in theory at least, be predicted if initial conditions are known in sufficient detail. Chaotic phenomena are very common in nature and can be detected as such, thanks to appropriate mathematical tools.
- **Attractors** are places, in a space of various variables defining a system (variable space), to which a dynamic system tends to drift. Attractors form around stationary states (see below).
- **Fractals** are mathematical tools, which allow for an analytical description of the borders or forms of multidimensional systems or bodies.

- ● *Singularities* are bifurcation points in dynamic functions, where the system jumps from one state to another without passing through intermediate points between the two states. Singularities define the non-continuous parts of non-linear systems.
- ● *Catastrophe theory* portends to develop a mathematical formalism, which allows the study of certain non-linear and non-continuous functions, which serve to describe some natural events before and after catastrophes, or radical non-continuous changes, occur.

Such mathematical descriptors, however, are applicable to a limited number of particular cases, and are not of general value for evolutionary biology. These mathematical tools are widely available today and have provided a fertile ground for several specific biological applications. I will not explore them further here, but will refer superficially to some of them occasionally (for a deeper analysis of these aspects see for example, Brooks and Wiley 1990, De Santis 1990, Kauffman 1993).

Thermodynamics and statistical mechanics as a science for complexity

If we accept that the study of biological evolution deals with complex systems, we could profit from the tools developed by other sciences dealing with complex systems. The science, which has dealt most successfully, for a long time, with complex interactions or with many body problems, is thermodynamics. The way thermodynamics has solved the problem of handling complex interactions is ignoring specific interactions and focusing on the characteristics of the whole system (state variables). This approach is also called the black box approach. One of the most important contributions of thermodynamics is probably the concept of state variables (such as temperature and entropy). A state variable defines the state of a system and is independent of the microscopic fluctuations of the components of the system. State variables allow studying the macroscopic or global behavior of complex systems. Statistical mechanics, in contrast, attempt to rebuild thermodynamic properties from first principles, i.e. from individual interactions, using statistical theory to derive thermodynamic states. Once a system is known thermodynamically, a statistical mechanical approach may help in our understanding of how the microscopic interactions achieve the macroscopic thermodynamic properties.

Among the main gifts thermodynamics has made to science are the empirical ***laws of thermodynamics,*** the so-called first and second law of thermodynamics. These laws are empirical in the sense that they cannot be derived from first principles but are given only by human experience. Thus, a single counterexample of any of these empirical laws will invalidate the law. Among the laws of thermodynamics, two are very important to biology. The ***first law*** states that mass and energy cannot be created but can only be transformed. The ***second law*** states that any change or transfer of energy has a cost in either entropy or free energy. Yet thermodynamics of closed systems, also called classical or equilibrium thermodynamics, is not applicable to most biological problems. Living organisms are open systems (Schroedinger, 1960) and thus, it is the study of non-equilibrium thermodynamics (also called irreversible thermodynamics, thermodynamics of open systems, or far from equilibrium thermodynamics) that is relevant to the study of the living. Some of the concepts from irreversible thermodynamics, which seem to be useful for biology, are the following:

- ● *Thermodynamic equilibrium*. Is the state of a closed system (i.e. a system that does not exchange matter with its surroundings) when its energy exchange with the environment is balanced so that output − input = 0. All closed chemical systems eventually tend to equilibrium. In biology, thermodynamic equilibrium for a cell or an organism is equivalent to its death.
- ● *Irreversibility.* In complex systems, the likelihood of a given process to revert, following exactly the same path as the one, which took the system to its present state, is very unlikely. For example, a ball, which rolls down a hill, is very unlikely to roll uphill, and if a force makes it to roll uphill, it will very likely follow a different path. All living processes are irreversible in theory and in practice. For biology, unlike physics, time has an arrow or direction. It is impossible that biological evolution reverts itself in time. Any process for which the probability of change is different for the future than for the past is an irreversible process. In this sense all biological processes are irreversible. A well-known example is the "evolutionary reversal" allowing the

return of the antecessors of the cetaceans to the see. This adaptation to the water followed different evolutionary routes to the one that let sea animals become terrestrial. Many physiological, behavioral and morphological characters show the terrestrial past of mammals re-adapted to living in the water.

- **Stationary states.** Open systems, which suffer continuous fluxes of energy and/or matter, do not reach equilibrium states. But often, open dynamic systems reach states, which are stable relative to other states, and the systems tend to drive to these states. These states are called stationary and are characterized by minimal energy dissipation in respect to alternative states.
- **Entropy.** The difference between the free energy and total energy of a system is entropy. A useful analogy of entropy is the concept of amount of disorder. Therefore, negative entropy or **negentropy** is analogous to order. All systems, released from external constraints, tend to maximize entropy. That is another formulation of the second law of thermodynamics.
- **Dissipative systems.** Open systems are systems through which variable amounts of mass and energy passes through. These fluxes may produce useful work. If so, the work produced by these fluxes will produce, thanks to the laws of thermodynamics, increases in entropy of the surroundings. Thus, the systems are said to dissipate energy and/or entropy. All living systems are dissipative systems, as the degradation of energy contained in the food they consume allow organisms to live, grow and reproduce. As the degradation and capture of energy from ingested food is not 100% efficient, entropy will be dissipated into the system.

The dynamics of irreversible and open systems are characterized by non-linearity and non-continuity. Conceptual and mathematical tools to study such systems have been developed by thermodynamics (see Prigogine 1980 for example). These tools seem appropriate for the study of biological systems, which seem to have properties, which are similar to complex chemical systems, such as irreversible evolutionary histories, dissipative structures and energy – entropy attractors. If dynamic biological processes are just more complex dynamic chemical processes, we should be able to eventually formulate **empirical laws of biological evolution** which could include **the law of birth and death of organisms** (or the law for the expansion and extinction of populations), as all living beings appear through the reproductive effort of an ancestor and eventually die. Other candidate subjects in biology for a thermodynamic treatment could include adaptation, irreversibility of evolution or the negentropic nature of evolution.

Computer simulations

The engineering approach for solving complex problems is building models. The models may be either physical or computational. Physical models of evolution would correspond to ecological experiments of evolution. Although such experiments are possible and desirable, few of them have been carried out and most questions of evolutionary biology are probably not amenable to ecological experimentation.

Computational models have had an extraordinary success in engineering, physiology, meteorology, astronomy, economics, etc. Thus, there is no reason why computational models should not provide us with a wealth of opportunities to study biological evolution. New computers provide wider opportunities to simulate ever more complex evolutionary scenarios. An additional advantage of computer simulations is that by repeating the simulations numerous times, we might quantify the frequency of the different outcomes, thus allowing for a **quantitative probabilistic evolutionary theory**. Other advantages of computer simulations are that they can be tailored to specific real life problems, that they can simulated macro and micro-evolution, that they can be as complex as desired and may include, in theory at least, all relevant variables. Computer simulations also have their limits, as computers are limited in power and memory, and programmers are limited in ideas. Despite these limitations, as a better understanding of the evolutionary process requires the simultaneous study of many different variables, computer simulations seem to be the best tools available at the moment. Computer simulations may help to:

- Pinpoint the relevant variables and components of the evolutionary process
- Discern the relative importance of different variables and processes
- Define the limits of the possible
- Assess the most probable and robust evolutionary strategy in different environmental scenarios

One graphical example of the powers of computer simulations can be explained using the following figure:
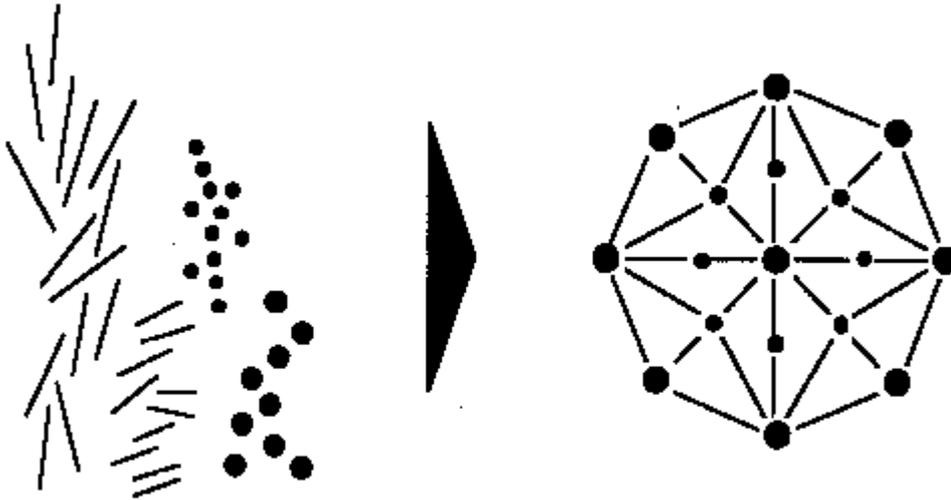
The properties of the rolling sphere in the figure can not be deduced from the component parts, unless the sphere is constructed from the parts and tested for its properties when rolling. That is what I mean when I refer to emergent properties of complex systems. Certain properties appear only after a certain degree of organization of the component parts has been achieved. Of course, other component parts can produce rolling spheres with similar properties. The same component parts can be assembled differently, producing similar rolling spheres. Thus, computer simulations are no proof for scientific theories, but they help us to decide what is possible, what could be missing, and most important of all, they help our minds form conceptual models of complex phenomena. In this sense, computer simulations are identical to all scientific theories. They are conceptual frameworks helping in our understanding of nature. Actual observations of nature and scientific experiments are always required to eventually falsify the theories and/or to improve them.

Ever more computer simulation models appear in the recent scientific literature and ever more evolutionary problems are being tackled with those simulations. In the next chapters, I portend to show how computer simulations, together with simple thermodynamic concepts, can expand our understanding of biological evolution.

4 The Model Biodynamica

ABSTRACT: In order to study evolutionary processes without having to assume too many simplifications, a computer simulation model called Biodynamica was developed. Biodynamica is a stochastic model for the study of the population dynamics of multi-loci agents. The program models a given number of individuals or virtual agents, each expressing behaviors according to the information coded in their genome which consist of up to 20 different genes or loci. These individuals form a virtual population and grow, breed and reproduce under various simulated environmental constraints. The model includes elements taken from Artificial Life, Evolutionary Programming, Genetic Algorithms, Bottom-Up Programming, Multi-Agent Programming, Artificial Intelligence and others and tries to keep simplifications of biological complexity to a minimum. Biodynamica allows for more realistic studies of the dynamics of biological evolution. In this complex simulation model, certain properties of the evolutionary process emerge naturally.

Biodynamica is freely available.

It can be downloaded, with its respective help files, from the Web at: http://atta.labb.usb.ve/klaus/klaus.htm

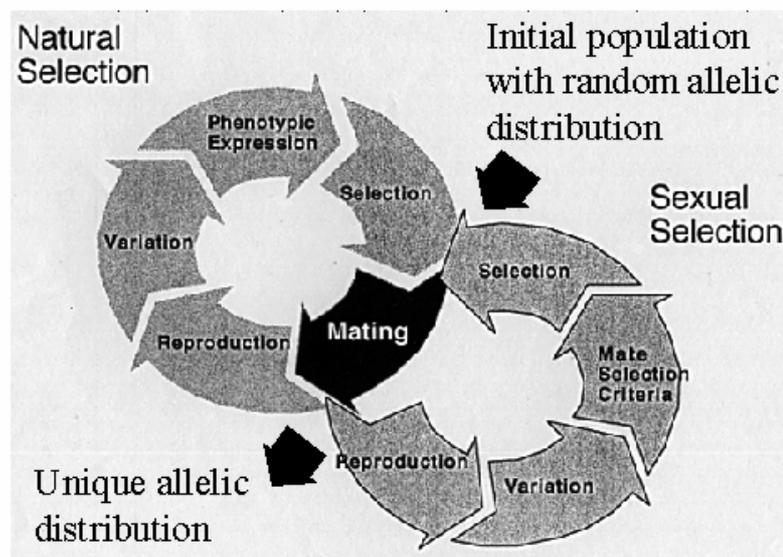## ADVANTAGES OF NUMERICAL SIMULATIONS

1. Mathematical models, that study the adaptive process of a matrix to a "dynamic fitness landscape, are referred to by physicists as "spin glass problems" (Mezard et al., 1987). It has been shown that these problems are particularly reticent to analytical solutions and are impossible to study with analytical mathematical models (Sinai, 1982; Durrett, 1986; Epstein and Ruelle, 1989). Analytical linear models do not represent accurately the complex interaction that occur in nature, whereas agent based simulation of evolution promises to give a better approach to what is happening in biological evolution (Ruelle 1991; Kauffman 1993; Jaffe 1996; Jaffe et al 1997; Levin et al 1997). Because of these characteristics, numerical simulation models seem, at the moment (and may be for a long time, see Ruelle, 1991), the only alternative available to study such complex problems. A numerical stochastic multi-agent simulation model, called Biodynamica, was purposely constructed to facilitate study of biological evolution. Biodynamica was build in order to allow for the following:

● Explore the properties of some variables that may be suspect of affecting the evolutionary process.
● Define the limits and specific conditions under which adaptation may work.
● Assess the effect of various features of biological evolution on an evolving population of organisms.

Biodynamica allows for the study of complex evolutionary interactions between different loci in populations of organisms using different sexual reproductive systems, life history traits, mating systems, parental investment strategies, etc. The main difference of the present model to previous ones is its complexity. This model allows the study of the effect of various selection pressures acting simultaneously on up to 20 different genes, each with an average of 10 different alleles, evolving simultaneously in one or several populations of organisms. This model searches, using a five step evolutionary processes, the matrix of a^g possible allelic combinations in a finite population of n individuals; where a is the average number of alleles per gene and g the number of loci or genes.

1. Simulation of the complete evolutionary process is not possible with the available computational tools. Thus, certain parts of the process have to be simplified for modeling purposes. If the evolutionary process, from the genetic code χ to the phenotypic expression Φ is

represented as a complex function $\Phi = \Phi \left(\Sigma_i \Gamma_i (\chi) \right)$, so that the genetic expression of the code for each gene $\Gamma (\chi)$ is viewed as a process dependent mainly on the genetic code χ, we can simplify the function so that $\Phi = \Phi (\gamma)$. That is, we assume that selection acts mainly on organisms rather than on its parts and that the evolutionary process implies the search of optimal combinations of parts (genes γ), where each gene can vary among a finite possible set of alternative alleles rather than by a larger set of possible strings of code χ. The difference between $\Phi (\gamma)$ and $\Phi \left(\Sigma_i \Gamma_i (\chi) \right)$ is the search space for optimal allelic combinations to form organisms among the total possible. For $\Phi \left(\Sigma_i \Gamma_i (\chi) \right)$ the total search space is proportional to the total number of possible codes of finite maximal length powered by the total number of genes. This search space is practically infinite as there are not enough atoms in the universe to form a single DNA copy of all possible sequences χ. Even for $\Phi (\gamma)$, if we assume a mean number of possible alleles of 10 for the average gene, the search space will be larger than 10^{1000} for most organisms. Classical genetic algorithms model the process associated with the function $\Gamma (\chi)$ which is a metaphor for the evolution of a single gene or protein based on random mutations of single loci of the code χ and on the selection of the resulting phenotype (Kauffman 1993 for example). Here I want to focus the process $\Phi (\gamma)$, which is a metaphor for the evolution of organisms, assuming very simple organisms composed of up to 20 genes providing a finite search space of 10^{20}. This metaphor, although still far simpler than real evolution, is significantly more complex than those assumed in models of genetic algorithms or of artificial life. Due to the complexities of the present model, it had to be validated in order to have some confidence in its results. This was done by producing predictions related to the odds for the emergence to genetic resistance to pesticides in sexual and asexual organisms, an area where sufficient experimental data is available. The results showed that the increased complexity of the model paid off, as its predictions where more accurate and less contradictory than those of alternative models in explaining the available experimental data (Jaffe *et al.* 1997).

Figure 4-1: Schematic representation of the model showing how, through the interaction between natural and sexual selection, populations of virtual individuals are driven to unique allelic distributions characterizing the various genes defining the organisms.

The advantages of using models like Biodynamica

The use of agent-based simulations allows new properties to emerge from complex systems. As we do not know in advance what the effect of interactions between numerous different variables will be, such simulations allow for stable states, stationary states, attractors, singularities, bifurcation points, and most of the features described in Chapters 2 and 3, to appear spontaneously in the simulations. These properties emerge without the need for establishing *a priori* any specific interaction pattern between genes. The model just tries to simulate as closely as possible real biological situations and through experimentation (i.e. large number of simulations exploring the parameter space) the existence of non-linear emergent properties will become evident by analyzing statistically the results. Each simulation is equivalent to a evolutionary experiment. By running multiple simulations, the probability of occurrence for specific evolutionary outcomes can be calculated.

Simulations can thus explore the outcome of evolutionary processes where various genes evolve at the same time (defined by the ***internal parameters*** of Biodynamica). By allowing different variances for different alleles of specific genes we may explore the possible parameter landscape of a given set of variables. For example, we may study the interaction between the evolution of live span and clutch size, or between mate selection strategies and those of sexual reproductive systems, or between all four of them, and assess the most probable evolutionary solutions of these interactions.

Variable environment guiding the evolutionary process may be simulated using rough complex dynamic fitness landscapes. Such ever-changing fitness landscapes can be produces (using different values for the ***external parameters*** of Biodynamica) by, for example, programming the occurrence of random variations of external parameters during the simulations. For example, optimal population sizes and the presence and strength of biocides can be varies randomly during a given simulation.

THE MODEL BIODYNAMICA

1. The numerical Monte Carlo simulation model Biodynamica consisted of a population matrix $M(i,j,k)$, representing a given number of organisms **i**, with gene loci **j**, possessing the allele **k**. At each time step, representing a reproductive cycle, each organism "**i**" was subjected to specific transformation rules defined by logical algorithms simulating the 5 transformation steps described below. For example a female individual $i=n$ of species 1 was referred to as $M(n,0,1)=1$ and $M(n,6,1)=1$ where $j=0$ indicates the gene defining the species and $j=6$ defines the gene coding for sex. This individual, in order to reproduce, had to find a mate $i=m$ such that $M(m,0,1)=1$ and $M(m,6,1)=2$. Always allele $k=1$ was expressed phenotypically, whereas allele $k=2$ was absent in haploids and in diploids was never expressed phenotypically but could be inherited and eventually expressed as allele $k=1$ in the offspring. The decision in diploids of which of the inherited alleles was assigned to $k=1$ and which to $k=2$ was performed at random at the conception (virtual birth) of the organisms (unless genetic dominance was simulated).

2. The genetic composition of the virtual population was monitored continuously, visualizing the dynamic properties of a complex interacting assemblage of genes evolving together in a population of organisms. An initial population of a given size (initial number of organisms) was created by randomly assigning in the matrix $M(i,j,k)$ to each individual i a specific set of genetic characteristics (alleles), corresponding to 20 different genes, each codifying for a specific phenotypic character. This initial population was then subjected to a five- step transformation program consisting of: Mate selection, Reproduction, Variation, Phenotypic expression, and Selection of the phenotypes.

The surviving population continued to endure the five-step transformation, at each time step. The exact genetic composition of the population was plotted at each time step, using the actual number of surviving individuals. Thus, the model allows assessing when and if the population reached a stable and/or optimal genetic composition. Optimal genetic compositions where those that allowed for a combination of phenotypes that in turn allowed the achievement of maximal population sizes. The speed of stabilization or the number of time steps needed for a population to reach the optimal and/or a stable genetic composition was used as an additional criterion to assess the viability of a given reproductive system.

Internal or biological parameters were initially assigned at random among the individuals of a virtual population, and then varied (i.e., suffered variation and selection) according to the constraints of the five-step transformation process. The internal parameters were modeled as a distinct gene coding for specific phenotypic characteristics of the organisms subjected to selection (see Table 4-1).

Table 1: Codes used for allelic expression for the various loci

j	Loci for:	Alleles code for:
0	**Species**	Different alleles define different species. No breeding between species
1	**Neutral gene**	Have no effect on the fitness of the organism
2	**Life span**	Maximum possible life span (in number of time steps) of the individual. For example, and individual having the allele 5 in loci 2 will have an maximum life span of 5 time steps.
3	**Clutch size**	Maximum possible number of offspring females may bear at each reproductive act. Actual clutch size will be constraint by life history parameters.
4	**Reproductive age for females**	Age at which females start reproducing.
5	**Sex ratio at birth**	Alleles with value 1 to 10 produce differently biased random female/male ratio, according to the allele in j = 14 (g14).
6	**Sex**	Defines the sex of individuals. Allele 2 produces males and allele 1 females.
7	**Reproductive strategy**	Allele 0 produces Asexuals, 1: Monosexuals, 2: Bisexuals, 3: Trisexuals, 4: Sexual-Asexual (as in haplodiploidy), 5: Sexual hermaphrodites (which mate only with other hermaphrodites), 6: Sexual hermaphrodites (which mate with females or hermaphrodites)
8	**Ploidy**	Alleles 1, 2 and 3 code for haploids, diploids and triploids respectively.
9	**Mutation probability**	Induce mutation rates at probabilities according to formula: $p = 0.2 \wedge (\text{allele} / 2 + 1)$.
10	**Resistance 1**	Resistance to biocide 1 in a continuous range; allele 0 is resistant and allele 10 not resistant
11	**Resistance 2**	Resistance to biocide 2 so that only allele 0 is resistant to biocide 2

12	**Resistance 3**	Idem gene 11 but for biocide 3
13	**Sex appeal**	Males with larger values of these alleles are sexier.
14	**Sex determination**	Sex is determined: 0= randomly in a 1/1 ratio. Gene 5 (g5) is neutral 1 = randomly but biased according to allele in g5 using algorithm rnd > g5 / allele in g5 then female, else male. 2 = by gene 6, following random recombination of alleles. G5 is neutral 3 = by meiosis of females g6. G5 is neutral 4 = by meiosis of male g6. G5 is neutral 5 = according to environment (values of Biocide 1) and g5 following algorithm g5 x 10 x pe1 x rnd < rnd then female else male 6 = according to environment (values of Biocide 1) and g5 following algorithm g5 x 10 x pe1 x rnd > rnd then female else male.
15	**Reproductive age for males**	Alleles define the age for reproductive maturity of males.
16	**Parental investment** Sex realizing the investment is defined by "External parameters"	0: No parental investment and no cost to fitness of parent 1: Communal care of offspring: Parent will invest equally in all newborns at a cost to its own fitness. Fitness of offspring = mean(g17)/10 2: Open parents: Parent will invest in their offspring and in others at a cost to its own fitness. Fitness of offspring = (mean(g17) + value of g17 of parent) / 20 3: Closed but opportunistic parents: Parent invest in own offspring, but without investing in others, at a cost to its own fitness. Fitness of offspring as in 2, but the parent's value of gene 17 do not contribute to the mean. 4: Closed and exclusive parents: Parent invest in own offspring without investing in others, at a cost to its own fitness. Similar to 3 but fitness of offspring = value of g17 of parent/10
17	**Degree of parental investment**	Amount of fitness increase or reduction the parent will provide to its offspring and its cost to the parent providing the investment. benefit = g17/10, cost = (g17/10) x cost of parental investment.
18	**Criteria for mate selection** **CMS**	0 (or > 13): Random selection of mates. Female mates with the first male encountered if it is an adult of its species (see gene 15) 1: Sexual Selection: prefers males with high sex appeal, i.e. high values of alleles in gene 13 2: Handicap: prefers males with high values in g20 3: prefers males with high fitness (see phenotype) 4: 1 gene: prefers males with low values in g20 5: 2 genes: prefers phenotypes 0 of g11 or g12 6: 3 genes: prefers mates with resistant alleles in g10, g11and g12. 7: prefers young males 8: prefers old males 9: Assortative mating, prefers males similar to her in genetic composition, except g1 and g6 (g18 included): 10: Dissortative mating, prefers males different to her in genetic composition (g18 not included) 11: prefers males similar to her in genetic composition (as g9 but g18 not included) 12: prefers males with the same allele in g18 13: prefers males with the same allele in gene indicated by g20
19	**Mating efficiency**	Number of males (or females in hermaphrodites) screened for mating according to criteria defined by g18.
20	**Free**	Neutral unless special conditions are simulated (see above)

The external or ecological parameters were set before the simulation and remained constant during a simulation. These were: optimal size of the population (or size above which density dependent selection increased exponentially, ops), initial size of the population (ino), number of genes (ng), proportion of individuals randomly killed at each time step (density independent selection), proportion killed if not possessing the correct alleles for resistance (gene dependent selection or biocide with strength pe1, pe2 and pe3), optimal clutch sizes (or clutch size above which fitness of offspring is reduced, ocs) and optimal age for reproduction (or age before and after which reproduction will decrease the fitness of mother and offspring, oar). In some simulations, some internal parameters where fixed, assigning all individuals the same allele and could thus be considered to be externally fixed. For example the mutation rate, number of individuals (males and females) screened for potential mates before mating (mef), and reproductive system (monosexual, bisexual or trisexual; and haploid, diploid or triploid) were often fixed with a single allele for certain simulations.

The five transformation steps are:

1. Mate selection: Females choose a mate so that mates were of the same species and of different sex, whereas hermaphrodite mated with a conspecific female or hermaphrodite, all according to mate selection criteria (MSC) defined by the allelic value of gene 18 (Table 1). The mate search was limited by the value of the allele of gene 19 (Table I). Females not finding the appropriate male mated with the one closest to the criteria searched for. If no conspecific male was found, the female did not reproduce during that time step or reproduced monosexually if hermaphrodite. When simulating trisexuals, one mate screened for the two other mates before mating.

2. Reproduction: Two different dimensions of sex were used: organismic sex or plurisexuality and genetic sex or multisexuality (or ploidy). These two dimensions defined the various possible reproductive systems as so that for asexual reproductive systems for example, populations possess only one sexual form of individuals (monosexuals) and individuals transmit a complete and single copy of genetic information to their offspring (cloning of haploids). Multisexual reproductive systems, modeled as polyploidy refer to sex at the level of the genome. Poliploid individuals possess more than one copy of genetic information and suffer recombination between the genes of these two copies when they reproduce. In multisexual reproduction, modeling sex at the level of the organism, only one copy of the alleles in each loci of the parent is passed on to their offspring. Plurisexual reproductive systems are those systems in which populations are composed of individuals of more than one sexual morph. Species could be monosexual (one sex), bisexual (two sexual morphs, i.e., females and males) or trisexual (three different sexual forms must meet and interchange genes before reproduction can take place). In addition species could be haploid (each individual possesses only one allele of each gene), diploid (individuals carry two alleles for each gene) or triploid (three alleles for each gene). Asexuals, under this nomenclature, are equivalent to monosexual-haploids.

Mated individuals produced offspring according to their phenotypically fixed clutch size (dependent on their allele for clutch size, their age and the size of the clutch their were born into), transmitting their genes to the offspring according to the following rules:

Asexual reproduction: genes of newborn were identical to those of the single parent.

Monosexual reproduction: only one sexual morph exists. Random reshuffling of alleles of genes (Recombination) of the single parent before production of newborn was possible only in di- and triploids.
Bisexual reproduction: each parent provided half of the genes to the newborn.

Trisexual reproduction: each parent provided 1/3 of its genes to the newborn.

Multi-sexual reproduction was possible in di- and triploids. In haploid organisms only a single copy of

each gene was present in each individual.

Diploid and *triploid* individuals had two and three copies of different alleles for each gene respectively. The proportion of genes transferred by parents to offspring was determined, according to the particular reproductive strategy of the female, following the rules of segregation (i.e. meiosis). That is, if the first allele of a given gene in the offspring came from the father, the second had to come from the mother, or vice versa. The specific allele of the parent being transmitted to the newborn was chosen at random.

Hermaphrodites reproduce as monosexuals or as bisexuals when they reproduce sexually.

When *haplodiploidy* was simulated, females transmitted their alleles to the offspring according to their ploidy number and their inheritance mechanism (either SS or SAS). Each parent provided at random alleles to the newborn with the probabilities indicated in Table 4-II.

Table 4-II: Probability for inheritance of at least one allele:

Inheritance mechanism	SS	SS	SAS	SAS
Ploidy number: Females	1	2	1	2
Males	1 2	1 1		
Maternal alleles to daughter:	0.5	1	0.5	1
Maternal alleles to son:	0.5	1	1	1
Paternal alleles to daughter:	0.5	1	0.5	1
Paternal alleles to son:	0.5	1	0	0

3. Variation: Randomly chosen genes mutated, changing their allelic value with a genetically determined probability (mutation probability), or at a fixed rate if gene 9 was programmed to have no variance (i.e. all alleles had the same value). The existence of genes determining mutation rates has been proposed (LeClerc et al. 1996 for example) and can be simulated here.

4. Phenotypic expression: A single allele (k = 1) was expressed phenotypically. The value of k for each inherited allele was assigned randomly for each offspring at birth. That is, Mendelian allele inactivation was simulated, where only one randomly selected allele per diploid loci was expressed phenotypically. That is, only one allele chosen at random was expressed phenotypically in diploids, whereas the only allele present was expressed in haploids. The phenotype expressed depended on the value of the allele as indicated in Table 4-I. Complex life history traits can be simulated. As an example, I present three simple features that were partially determined by three genes, similar to what is known to be common among animals (Roff 1992): Maximal life span, maximal clutch size, and age of first reproduction. The final clutch size of each individual was calculated based on the allelic characteristic of the gene coding for clutch size and the age of the reproducing individual, using a normal distribution, so that:

$$cs(i) = M(i,3,l) / [(oar^{-0.5}) ^{-\{ [(age(i)-oar)^2] / oar\}}] \ .... (1)$$

Where $cs(i)$ is the clutch size of individual i, $M(i,3,1)$ is the allelic value for maximum clutch size in the phenotypically expressed genome of individual i, $age(i)$ is the age of individual i, oar is optimal age for reproduction fixed as an external parameter in each simulation. That is, the maximal clutch sizes were partially predetermined genetically and occurred at an optimal age of reproduction. Clutch size affected the probabilities of survival of the future adult, so that individuals born in clutches larger than optimal decreased their probability of survival exponentially. The equation here was:

$$if \ cs(i) <= ocs \ then \ fit(k) = 1 \quad else \ fit(k) = ocs/cs(i)^3 \ .... (2)$$

Where cs(i) is the size of the clutch in which individual k was born, ocs is optimal clutch size fixed as an external parameter in each simulation, and 1/fit(k) is the additional probability for the offspring k being eliminated by random selection (see below).

Selection: The model did not assume any simplified expression of fitness but reproduction and individual survival were decomposed into different aspects for selection to act. Individuals were excluded from the population when any of the following criteria applied:

1- Their age exceeded their genetically prefixed life span.

2- When randomly selected with a probability which increased with population density and with the value 1/fit(i) in equation 2 using the algorithm:

if rnd > ops/n x 1/fit then the individual was killed

3- When parents clutch size exceeded optimal sizes at high population densities.
- When a biocide was applied to randomly trim the population from individuals not possessing the right resistant phenotype: Individuals with non resistant phenotypes of genes 6 and 7 in Table 4-I were killed randomly with probabilities which varied randomly from 0 to 0.95 according to values of pe1, pe2 and pe3.

The population endured the five-step transformation, at each time step, representing one reproductive cycle. Generations overlapped as each female reproduced independently. Thus, one time step corresponded, under the life history traits simulated here, approximately to 0.2 generations (i.e. 5 time steps corresponded in average to one generation). The exact genetic composition of the population was plotted at each time step, with the actual number of surviving individuals. Thus, we may assess when and if the population reached a stable population size, and at the same time monitor the genetic composition of the population at each time step. Optimal size of populations was 400 and the initial size of the populations was 200 individuals, unless stated otherwise.

5. Natural Selection: Individuals not able to reproduce or to survive environmental chaleges did not transmit genes to the next generations.

Quantitative analysis of probabilities:

A successful genetic reproductive strategy in micro- and macro-evolutionary terms has to fulfill at least three basic criteria, covering various levels of selection (Williams 1992; Wilson and Soler 1994):

1- Alleles coding for the strategy have to be able to displace other competing alleles coding for different strategies, and once fixed in a population, they have to stop other alleles, coding for rival strategies, from re-invading the population. This is called an Evolutionary Stable Strategy (Maynard-Smith 1976).

2- The strategy has to allow organisms and populations to follow an evolutionary dynamic which can adapt successfully to changing environmental challenges (i.e. to a rough fitness landscape). That is, it has to be adaptive in the short term. i.e. has to have high classical Darwinian fitness (Dobzhansky 1951).

3- The strategy has to allow organisms and populations to maintain a healthy genetic variability, providing the population with a sufficiently large allelic assortment for eventual future environmental challenges. That is, it has to be adaptive in the long term, i.e. have long term species feasibility or species stability (Gould and Lewontin 1979, Sepkoski 1993).

To test for these possibilities, three types of experiments were performed:

1- Simulation of "*gene selection*" aimed at assessing the effect of selection on the allelic distribution of a

given gene in a single interbreeding population. For example, alleles coding for different parental investment strategies (or for different sexual strategies) were assigned at random but uniformly among the first individuals. The simulation was started and the frequency in the population of the various alleles coding for a specific strategy was monitored during the simulation for each time step. In simulations designed to assess the stability of certain sexual strategies (i.e. assessing if they where ***Evolutionary Stable Strategies*** for example) , an initial population with a given strategy was created, and after a given number of time steps (normally 20), one or a few mutants with a different strategy were introduced and the frequency of these alleles was monitored for at least a further 400 time steps or until the mutant allele displaced the other.

2- In simulations of "***group selection***", several non-interbreeding populations (i.e. different species), each possessing a specific reproductive system, were made to compete for the same computer memory and time (i.e., maximum population size of the sum of all species was limited), and the growth of the population of each species was monitored. The initial populations for each species were created independently, each with a random distribution of the genetic characteristics of individuals, except those that were unique for each species. The population displacing the others was considered to have survived an evolutionary process. Repetition of this kind of simulation, with different random initialization, provided the basis for calculating the probability of survival of the population of a given species. In certain cases, the probability of finding a right mate before reproducing (i.e., the cost of mate selection in reproduction) was manipulated so that it was the same for all reproductive systems tested.

3- Survival probabilities of "***isolated populations***" were assessed by randomly creating initial populations in which all individuals had one common characteristic (i.e., the same reproductive strategy), and evaluating the probabilities of these populations to survive a variable environment for a given number of time steps.

Each experiment was repeated for at least 200 times and until standard deviation was less than 20 % of the mean. Relative probabilities thus provided a quantitative assessment of which strategy was more likely to be maintained or to emerge during the evolutionary process.

5 On mono-, bi-, and tri-sexuals

Adapted from: Jaffe 1996

ABSTRACT: Darwinian evolution is normally modeled as the outcome of the interaction between reproduction, variation and selection. These interactions may become very complex, especially when considering that selection may act on several genes simultaneously. The results from simulations with Biodynamica presented here emphasize that biological evolution is an irreversible process showing strong genetic drift. Simulations of "gene selection" showed that asexual reproductive strategies were very stable in evolutionary competitions and consistently out-bred sexual ones. Bisexuality succeeded in replacing asexuality only in simulations of "group selection" with changing environments and could be maintained only under group selection if non-random mate selection mechanisms were at work. Other simulations showed that in trisexual organisms, mate selection mechanisms and natural selection do not work efficiently. Thus, reproductive strategies with more than two sexes are improbable. Polyploidy may improve evolutionary success among monosexuals as it reduces the exhaustion of the variability of the gene pool due to continuous selection pressure. I postulate that sex evolved under changing selection pressures, allowing the emergence of sexual selection, which allowed sex to stabilize, thanks to the strong irreversible dynamics in the evolution of complex organisms.

Why the sexes are, in fact, always two?

Fisher (1930) once commented: "No practical biologist interested in sexual reproduction would be led to work out the detailed consequences experienced by organisms having three or more sexes; yet what else should he do if he wishes to understand why the sexes are, in fact, always two?". In spite of appearing to be a simple and fundamental question for biologists, this question has until now eluded an answer (e.g. Maynard Smith, 1978; Margulis and Sagan, 1986; Greenwood and Adams, 1987; Stearns, 1987; Hamilton et al., 1990; Williams, 1992; Heitkoetter and Beasley, 1994; Judson and Normak, 1996; Hurst and Peck, 1996). To have a deeper understanding of the working of the evolutionary processes, it is unavoidable to address this question. To do so, I have defined operationally what "more than two sexes" may mean. Most recent definitions use the control of inheritance of cytoplasmic genes as a criterion to define the sexes (see Hoekstra 1987, Hurst and Hamilton 1992). Here I analyze the dynamics of the various sexual reproductive strategies rather than the sexes. I explore the limits and specific conditions in which sex may evolve, i.e., assess the effect of sex on a continuously evolving population of organisms and compare it to asexual alternatives using a numerical simulation model. Finally, I compare the theoretical results obtained (i.e., the emergent properties of the model) with real life and make concrete predictions which can eventually be falsified by experiments.

Defining sex:

Multi- and pluri-sexuality may mean two different things:

1-More than one copy of genetic information is kept in the organism or cell; I will call this multi-sexuality. These copies may reshuffle to produce a new combination of genes or alleles in the organism to be born (meiosis or molecular sex). This can be achieved theoretically by different means and mechanisms, but in living organisms, diploidy and polyploidy seem to be the most common mechanism used. This kind of sex is thought to accelerate evolution as it increases genetic variability (Maynard-Smith, 1978; Ruelle, 1991; for example).

2- Mating with one or more mates (here referred to as pluri-sexuality) before each reproductive act, where each mate supplies the new organism with part of its genetic information (mating or organismic sex). This kind of sex is thought to increase the genetic variability in a population, through mixing of genes from different individuals, when mutation rates are lowered (Eigen and Schuster, 1979; Hamilton et al., 1990;

for example). This mechanism also allows the transfer of genetic information, acquired by a few individuals, to the whole population (Dawkins, 1986).

General dynamic properties:

Using simulations with organisms defined by 15 different genes (see end of chapter), some general dynamic properties emerged from the visualization of the changing allelic composition of the evolving populations. Neutral genes in organisms with multiple additional genes subjected to gene selection showed a strong genetic drift even in large populations (Demo01 and Demo02 of Biodynamica). The dynamics in the allelic frequency distribution of neutral genes was not very different from that of genes suffering selection pressure (clutch size, age for reproduction, for example). Alleles of neutral genes maintained their random distribution in a population only if the organisms suffered low selection pressure. At high selection pressures, neutral genes showed strong genetic drift (Demo03 and Demo04). Strong selection pressures on other genes affected even genes that had a small effect on the final phenotypic expression of the organism. For example, genes for resistance, when selection for these genes was low, showed strong genetic drift when the population was adapting genes with a strong effect on survival and reproduction probabilities such as life span and clutch size. The final frequency distribution of alleles from all type of genes was less predictable as the number of genes subjected to selection grew larger, and was sensitive to initial conditions (i.e., initial composition of M(i,j,k) see Demo05 in Biodynamica). The genetic characteristics of a population, once evolved, were not easily reversed by reversing selection pressure, demonstrating the irreversible dynamics of this evolutionary process. That is, the genetic structure of the population was affected by past selective pressures and thus, the population as a whole was characterized by their adaptive history.

The resilience of asexual reproduction:

Populations stabilized their allelic composition after about 20 time steps. The variance of various genes in different populations was averaged using several simulations. Fig 5-1 and 5-2 presents the average variance of neutral genes and that of resistance genes in "Isolated populations" with a specific reproductive strategy. Asexual populations showed that selective pressure reduced or exhausted the variability of the gene pool, that is, they always showed a low variability of the gene pool independently of population size. Monosexual populations, if polyploid, maintained high genetic variability, similar to that shown by bi- or tri-sexuals. Bisexual and/or bi- and polyploid populations maintained a larger variability of their gene pool, even after stabilizing the genetic frequency distribution (Demo05 to Demo07). Diploid and triploid populations, or haploid plurisexual populations were more resistant to the depletion of the variability of the gene pool due to selection compared to asexual (monosexual haploids) organisms.

Populations that had an asexual reproductive strategy adapted faster to environmental constraints but consistently failed to stabilize the allelic frequency distribution at optimal values (Fig 5-3), unless mutation probabilities were very high (i.e., more than 10% of alleles mutated). Sexual populations responded much slower to selective pressure, but had a higher probability of reaching optimal genetic compositions. High fixed rates of mutation favored the survival of asexual populations compared to sexual ones. When mutation rates were subjected to selection, the various reproductive systems stabilized their mutation rates after an adaptive process of 30 time steps. The rates were statistically indistinguishable and stabilized at values of 0.002 mutations per genes per birth.

The probabilities of sexual alleles displacing asexual ones in an interbreeding multi-sexual populations, when simulation "gene selection" in a stable environment with several populations competing were practically nil as asexual populations nearly always out-bred the others (Table 5-I). Chances of finding a mate represented a hindrance for plurisexual organisms compared to monosexual ones. Thus, I modeled a situation in which the odds to find a mate were the same for all reproductive systems and in which asexuals were forced to produce only half their offspring, making them equal in output to sexual organisms. But even then, if a single population was allowed to freely mutate from one

reproductive system to another, asexual individuals out-breed all others. Sexual organisms out-bred asexual ones only during short time spans of the simulations and in special situations with external conditions similar to those tested by Hamilton et al. (1990), in which I simulated very large fluctuations among different selection pressures in small populations and/or where sexual organisms selected their mates based on their ability to resist parasites. As new mutations could produce asexual organisms again, these reinvaded the population. Thus, asexuality was an evolutionary stable strategy.

When sex is advantageous

When simulating selection acting on "Isolated populations", sexual organisms showed always a slightly higher survival rate than asexual ones (Table 5-I). It was only in competitions among non-interbreeding populations with different reproductive strategies simulating "Group selection", when bisexual populations outperformed asexual ones, and then only in competitive encounters where mate selection was not random (Fig 5-4 and 5-5). I used the assessment of resistance to parasites or illness (health) of mates as the basis for simulating mate selection. This criterion alone was enough for bisexuals to overwhelm other populations. More complex mate selection criteria, some including selection of females by males, improved the relative advantage of bisexuals. The selection of the appropriate criteria for mate selection, turned out to be a complex and important area of research in its own right. For example, excessively strong mate selection reduced the probability of survival of sexual populations. These complex aspects of mate selection will be dealt with elsewhere.

Why are the sexes always two

Trisexual organisms had several comparative disadvantages to bisexual ones. Populations with trisexual organisms could not compete with bisexuals. That is, mate selection did not work as well for trisexuals as for bisexuals (Fig 5-5). Adaptation of haploid-trisexual and triploid-plurisexual populations was very viscous, compared to bisexual-diploid or bisexual-haploid populations. Optimal genetic composition was reached at slower rates compared to bisexual populations (Fig. 5-3). In addition, the variability of the final gene pool was higher than for bisexuals for genes submitted to strong selection (Fig 5-5) suggesting again more difficulties in adapting to environmental constraints.

Some conditions for the emergence of sex:

Overall then, simulations suggest that asexual organisms were better prepared to adapt if external conditions were stable and if initial population sizes, mutation rates and growth rates were high. Sexual organisms had a definite advantage over asexual ones only if mate selection was present, if adaptation implied a fine tuning of a complex assemblage of genes to a changing environment, and if mutants for sex could evolve isolated from asexuals. The presence of non-random mate selection (sexual selection) gave bisexual organisms a clear evolutionary advantage over monosexual ones in an evolutionary context simulating group selection. Thus, in a strict sense, sex was not evolutionary stable, it was evolutionary feasible, as asexuality could invade again sexual populations due to the fact that advantageous genetic compositions selected through sex were transferred to asexual mutants.

Consequences for evolutionary theory

The results allow proposing a more realistic and complete dynamic evolutionary scenario. Neodarwinian theory assumes that biological evolution is based on all the five processes modeled here but models so far have concentrated on three fundamental aspects: Vertical transmission of genetic information, the introduction of novel variation through random mutation of genes, selection of organisms. Models have assumed further that biological evolution is a continuous process that allows a potential infinite variety of organisms to exist and adapt. However, we know that physical (mainly thermodynamic) laws restrict the dynamics of evolutionary processes. It is known that completely random shuffling of genes is not probable (Mayer, 1990) or possible (Kauffman, 1993). The interaction of genes in complex systems produces a complex dynamics that is not detectable when studying the adaptation of a

single or a few genes. The results here confirm those suppositions. They show the existence of strong dynamic interaction between simultaneously but independently evolving genes, suggesting a nonlinear, irreversible nature of the evolutionary process (Jaffe, 1980; Jaffe and Fonk, 1995). A "genetic pull", or "random drift" (Wallace, 1986), sometimes called the Hill & Robertson effect (Hill and Robertson, 1966; Felsenstein, 1974; Maynard-Smith, 1978), occurs when selection on alleles at one locus produce gene frequency changes at other loci. Here I suggest that this effect must be much stronger in naturally evolving populations of organisms than formerly recognized.

The fact that evolutionary dynamics is irreversible and that asexuality is an evolutionary stable strategy is acknowledged by Margulis and Sagan (1986) who wrote "(It) is not because sexual species are better equipped to handle the contingencies of a dynamically changing environment but because of a series of historical accidents (that sex exist)". Many ecological, physiological and biodynamical reasons why sex should not exist have been put forward (Mulcahy, 1975; Stearns, 1987; Greenwood and Adams, 1987; Maynard-Smith, 1978; Judson and Normak, 1996; for example). A convincing argument for the existence of sex, was put forward by Hamilton et al. (1990) based on the advantage of sexual organisms to adapt faster than their parasites. My results confirm that biodynamically, sex is difficult to justify if evolution is viewed as a three-step process. The five-step model suggests that the main evolutionary advantage of sex is that it allows for mate choice. Appropriate mate choice in turn provides a mechanism that can direct the future genetic variability of a population by selecting highly adaptive phenotypes (and thus indirectly genotypes) even if natural selection acts discontinuously, which allows sexual organisms, among other things, to escape their parasites. This restriction of random variation through sexual selection has at least two advantages: it may select organisms with the right genotype to challenge future natural selection pressures, and it provides a kind of direction to the evolutionary process by selecting organisms based on sexual selection criteria which may provide adaptive potentials not easily selected for by short term natural selection processes. Appropriate sexual selection criteria, thus, may accelerate evolution enormously by providing a new dimension to the evolutionary process, giving a definite advantage to sexual organisms over asexual ones. Although sex with random mating may have evolved under very exceptional conditions, it was probably only when by chance, the first efficient mate selection criteria evolved, that plurisexuality could be fixed by evolution. Because asexuality is evolutionary stable, the emergence of sex can be explained only if we assume a strong irreversible dynamics in evolution.

Non-random mating

Biology textbooks are filled with examples of refined mechanisms used by individuals to find their mates. Thus it seems reasonable that mate finding is not limiting in sexual organisms. The results of the model show that if we ignore the problem posed by mate finding, pluri-sexuality improves mixing of the gene pool in a population, whereas polyploidy improves the maintenance of genetic variability in a population, even if submitted to strong selective pressure. The original model of Fisher (1930) and Muller (1932) suggested that in the absence of recombination, an increase in the frequency of an allele at one locus completely prevents increases in frequency at another. This Muller's ratchet (Muller, 1964; Maynard-Smith, 1978) was thought to provide asexual organisms with a hindrance as they accumulated more deleterious genes than sexual ones. The model suggests that although the Muller's ratchet exists, it is the appearance of complex new recombinations, not attainable by random mutations, that ease fine adaptations to changing environments and that may give sexual organisms an eventual advantage over asexual ones.

The limits of sex

Some of the sexual reproductive systems simulated by the model, such as trisexuals, do not occur in nature. I think to have provided a rationale for this: too much plurisexuality or gene mixing makes mate selection difficult to achieve. Trisexual-triploids or species with "more than two sexes", in which sex A, B and C have to fuse are not known and its existence is not likely as this system does not add on genetic variability but becomes very viscous to adaptation. That is, the existence of putative trisexual population will not be likely because of the viscous behavior towards selection of such populations, due to the

decreased probability of three mates meeting compared to two, and because mate selection in trisexual populations is inefficient. Other alternatives, such as a system in which A can mate with B and C, B with A, etc., do exist and are in the end similar to a bisexual situation. Hurst and Hamilton (1992) studied such a situation, in which sex was defined according to the control of the inheritance of cytoplasmic genes, and found that true multiple sex in such a situation were either non-existent or binary. But "more than two sexes" may mean polyploid organisms or multiple-mating organisms, which do occur in nature. Maintaining a healthy genetic variability is important for biological species. This can be achieved more efficiently through multiple mating with a bisexual-diploid system rather than through a trisexual-triploid reproductive system. The results suggest that polyploid-trisexual organisms are unlikely to exist, providing a tentative answer to Fishers's original question stated above.

The enigma of excessive sex

Bisexual-haploid populations are known to exist (mosses and most fungi). The model suggests that they should represent one of the more successful sexual strategies. But many bisexual biological species are diploid, although phenotypic expression of the allele in the genotype of those organisms is not random. Dominant alleles will be expressed always if they are present. Thus, biological bisexual-diploids can probably be considered as intermediate between the simulated bisexual-haploids and bisexual-diploids, where no dominance in allelic expression was simulated. Regarding the fungi studied by Nauta and Hoekstra (1992), it seems that heterothallic fungi are equivalent to what I call bisexual-haploids; whereas homothallic fungi are intermediate to the bisexual-haploids and monosexual-diploids. Homothallism, at least during the sexual phase, involves two strings of genes, which although originated in different cells join to mix their genes. If the two cells are considered as different organisms, we may model the system as bisexual-haploids, if the two cells are regarded as from the same organism, the fact that two string of genes mix during reproduction is the dynamics equivalent of monosexual-diploids. In this respect, the model suggest that intergametophytic selfing is not completely equivalent to a pure asexual system as postulated by Nauta and Hoekstra (1992), but that homothallic fungi enjoy some advantages of sex.

Explaining polyploidy in plants

The model suggest that Monosexual species are better in fast tracking environmental changes of ecosystems and in tolerating high inter-specific competition. Thus, they should be common. But for monosexuals, polyploidy is relatively more advantageous than for bisexuals (Fig 5-1and 2). Thus, monosexuals are more likely to evolve polyploidy to avoid the exhaustion of their genetic variability due to strong and long selective pressure. I predict, for example, that among auto-compatible plant species, or among asexual reptiles, which could be considered as monosexual, polyploidy should be common. Or stated differently, polyploidy in plants should be negatively correlated to sophisticated sexual mating systems allowing for mate selection in plants. The existing literature shows that polyploidy in plants correlates strongly with monosexuality (Gustafsson 1946, Bierzychudek 1985, Asker and Jerling 1992, Hilu, 1993; Moody, 1993).

Asexual reproduction is so efficient that it occurs even among highly evolved complex organisms (Judson and Normak, 1996). This may explain why many insect species (for example most Homoptera) and many plant species reverted to facultative asexual reproduction using both asexual and bisexual reproductive strategies complementarily.

The importance of mate selection

Bisexual reproduction in the long term can only be maintained if mechanisms are in place that allow for a non-random selection of mates. I predict that mate selection among bisexual organisms should be very common (if not the norm) and should affect the genetic composition of the population. Testable predictions in this sense would be to expect sexual selection mechanisms to be at work among nematodes or among sexual fungi such as the filamentous ascomycetes studied by Nauta and Hoekstra (1992). If

mate selection is really such a basic mechanisms guaranteeing the working of sex, we should also expect it to be at work among humans. Population genetics and mate selection in humans is fairly well understood and thus specific predictions can be made. For example, female preferences (Jaffe and Chacon-P, 1985) for taller and more intelligent males (Buss, 1989) should drive the genetic composition of our species, partially explaining the continuous increase in average body size and IQ of modern human populations. If the reported continuous increase in average size and IQ among humans is even only partially driven by sexual selection, then I predict that the size and IQ of males should correlate with their fecundity. That is, on average (and taking other factors such as nutrition and health into account), size and/or IQ of non-married or childless males should be lower than that of their fecund fellows in societies which allow females to choose their mates (some indirect evidence to this may be found in Weinrich, 1977; Thiessen and Gregg 1980, Perusse, 1993). In general, the recent literature suggests that courtship and mate selection is not random and is strongly directed to increase the fitness of the offspring not only among humans (Grammer 1989, Voland and Engel 1990, Swaddle and Cuthill 1995, Weedekind et al. 1995), but also among birds (Owens and Short 1995) and other organisms. Any non random mate selection mechanism that increases the fitness of the offspring will in the end affect the genetic composition of the population and will accelerate evolution in the direction defined by the mate selection criteria, as predicted by the model.

Experimental parameters

Unless stated otherwise:

Internal or biological parameters were initially assigned at random among the individuals of a virtual population, and then varied (i.e., suffered variation and selection) according to the constraints of the five-step transformation process. Organisms possess 15 genes, each with 11 different allelic possibilities (0-10), except sex (2 possibilities). The internal parameters were modeled as a distinct gene coding for specific phenotypic characteristics of the organisms subjected to selection. These genes j were: species (where one allele defined the species and which in this case were immune to mutations), maximum life span (with 10 possible allelic values of k coding for life spans from 1 to 10 time steps), maximal clutch size (11 alleles coding for clutch sizes from 0 to 10), minimum age for initiating reproduction for males and for females separately (5 alleles each), mutation probability (11 alleles coding for mutation rates from 0 to 0.2 mutations per gene with logarithmic increments), mutation intensity (11 alleles coding for new mutated values close or far from the original value of the allele suffering mutation), sex (2), sex ratio of newborns (10), two kinds of resistance to external constraints (i.e., resistance to illness, draught, parasites, heat, etc., with 11 alleles each, where only allele 0 was resistant to that particular environmental challenge), reproductive strategy (if not fixed as an external variable) was coded by 2 genes, one for polyploidy (with 3 alleles, coding for halpo- diplo or triploidy) and one for multisexuality (3 alleles coding for mono- bi- and tri-sexuality), number of individuals (of any sex) screened in search of potential mates before mating (90), kind of parental investment (4), degree of parental investment (10), sex appeal (10) and two neutral genes (with 10 alleles each) whose phenotypic expression did not affect the fitness of the organism.

External parameters were set so that density independent selection eliminated each time step 1 out of each 100 individuals chosen at random. Individuals not expressing the resistant allele of gene R2 and R3 were killed with a probability $pe2 = pe3 = 0.2$. Optimal population size (=800), initial population size (=800), maximum number of individuals encountered before mating (=50), optimal clutch size (=4), optimal age for reproduction (=2), mates chosen at random.

Relevant Demos in Biodynamica: Demo01 to Demo07 and Demo10 to Demo15.

Figure 5-1: Average variance in allelic composition of neutral genes, present in the population after 30 time steps of simulations for "isolated populations", for various reproductive strategies (n > 200 simulations for each bar). External parameters were: number of genes = 9, ino = 500, ops = 1000, pe2 and pe3 varied randomly every time-step between 0 and 0.9, mef = 400, ocs = 4, oar = 2, mates were selected at random.
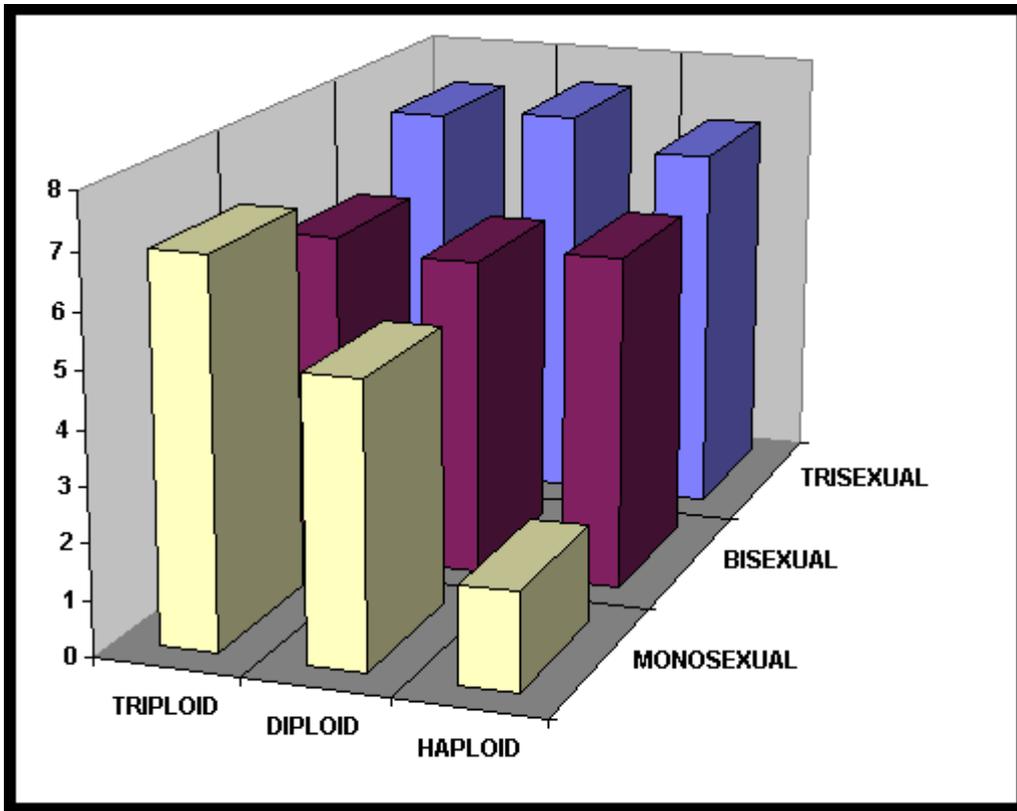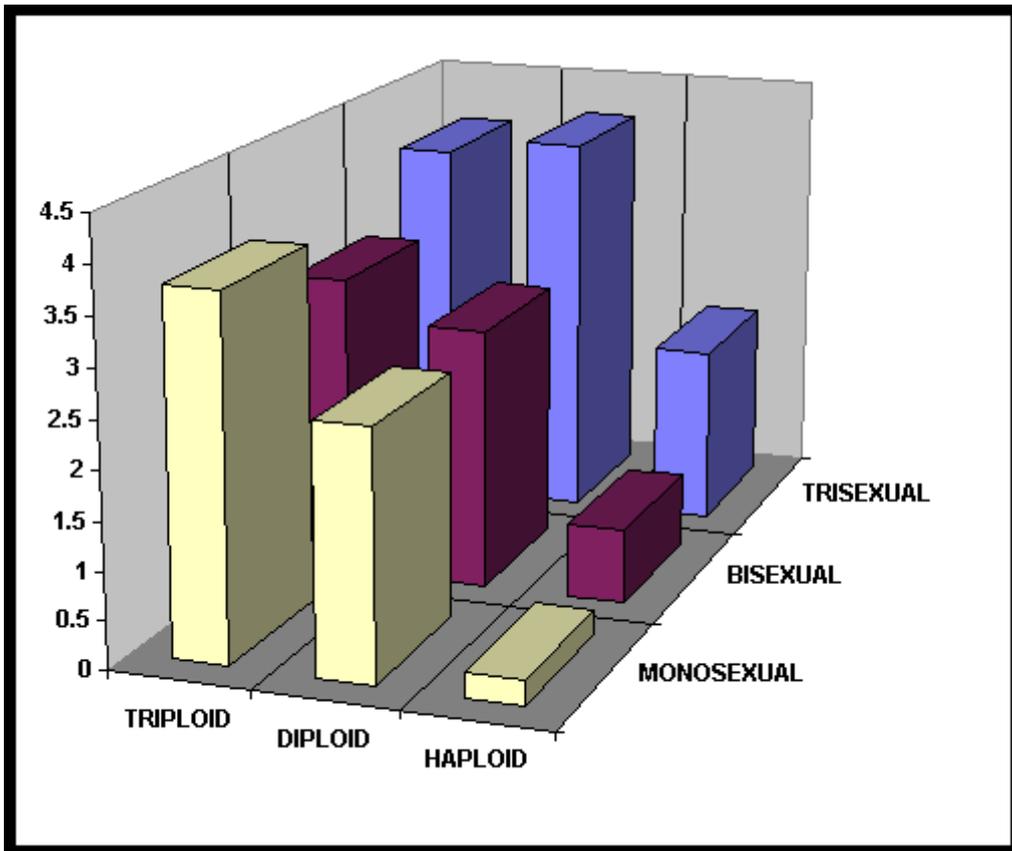

 Figure 5-2: Average variance in allelic composition of genes for resistance, present in the population after 30 time steps of simulations for "isolated populations", for various reproductive strategies (n > 200 simulations for each bar). Simulation constants as in Figure 5-1.

Figure 5-3: Number of organisms (as % of maximum possible) in randomly created populations of asexual (blue line), bisexual (green) and trisexual (red) haploids. Conditions of the simulation were those of Fig 5-1. Similar curves were obtained with monosexual haploids, diploids and triploids. The figure summarizes the findings: asexual population grow faster or evolve populations with high fitness faster compared to sexual ones, but they often do not stabilize at optimal genetic composition due to strong genetic drift and low genetic variance. Sexual populations evolve stable genetic compositions slower, but are more likely to reach optimal values.
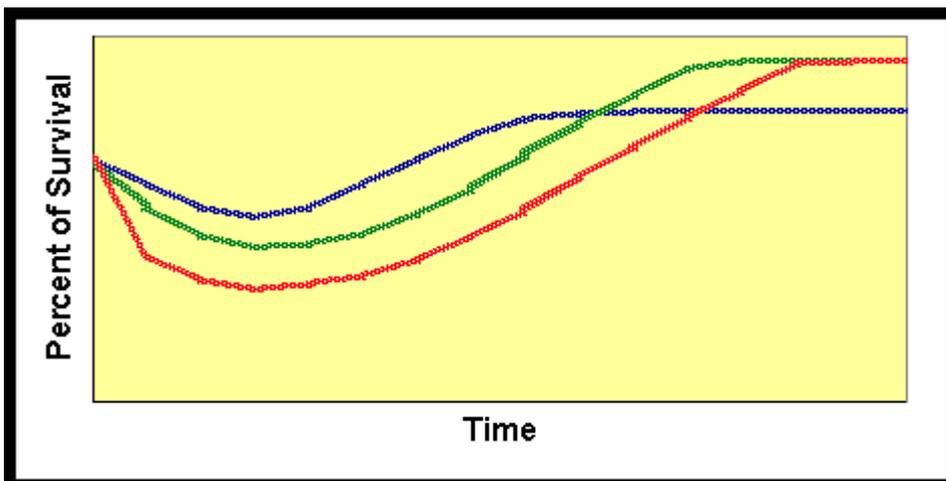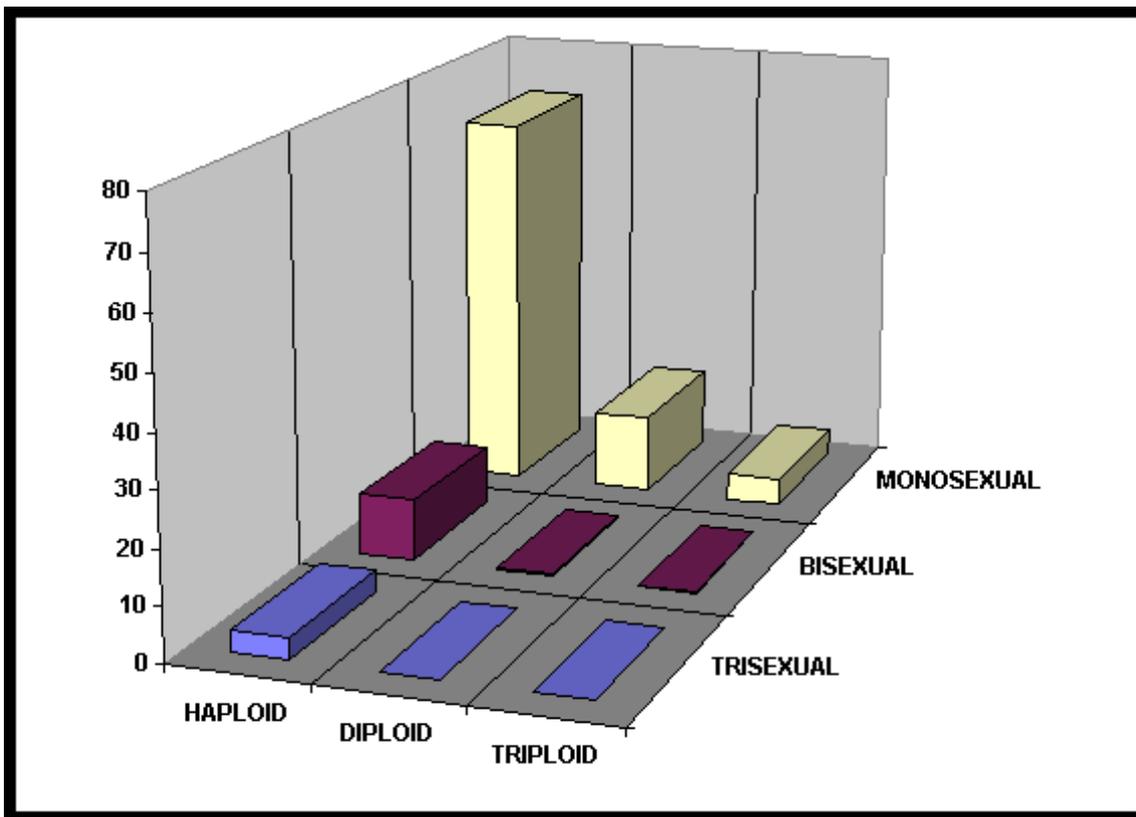

Figure 5-4: Probability (%) of survival of competing populations, created at random, possessing, different reproductive strategies emulating, Results are from "group selection", for organisms selecting mates at random (n = 1500 simulations, Demo14 with allele 0 for mate selection). External parameters of the simulation as in Fig. 5-1, except that ino = 300 for each population, ops for the total population was 2000, pe1 and pe2 varied randomly during the simulation (between 0 and 0.9).

Figure 5-5: Probability (%) of survival of competing populations, created at random, possessing, different reproductive strategies emulating. Results are from "group selection", where sexual organisms selected their mate based on health, i.e., phenotypic expression of R1 or R2 = 0 (n = 1500 simulations of Demo14 with allele 5 for mate selection). The external parameters of the simulation were as in Fig. 5-4.
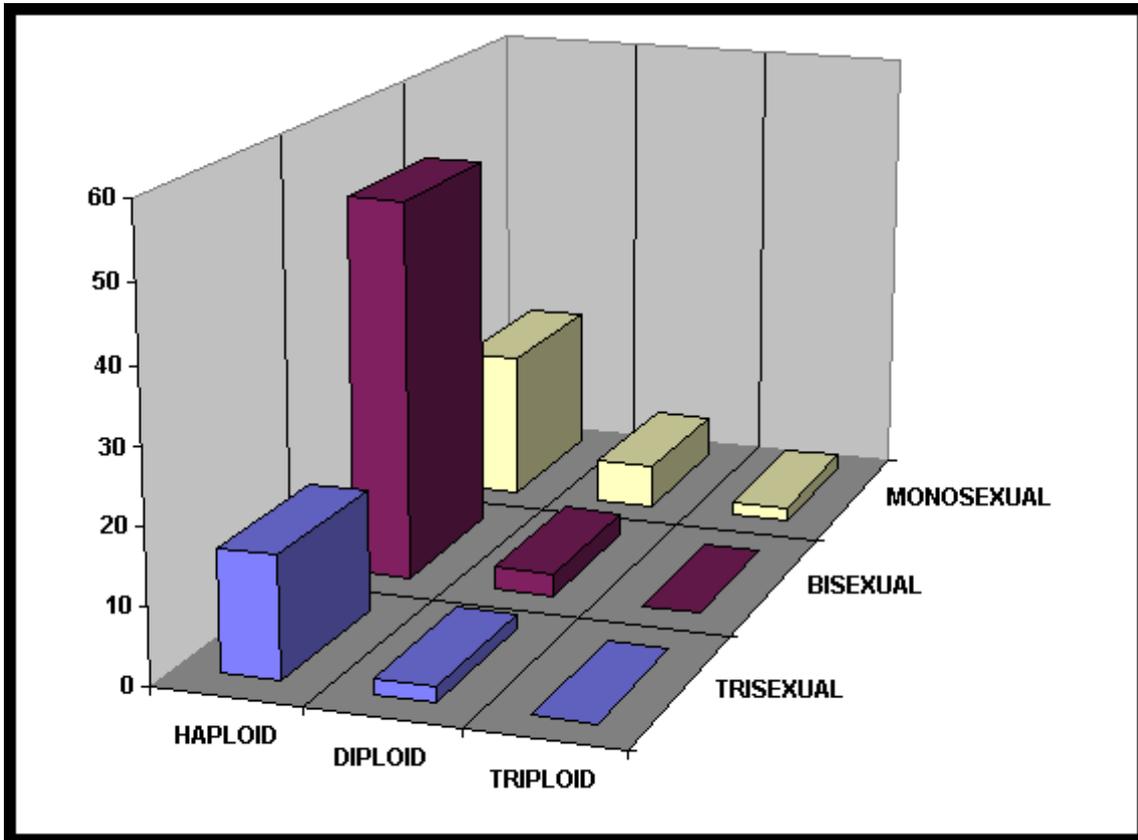

Table 5-I: Probabilities (%) of a given population or gene to survive or displace competing strategies successfully, in 20 time steps, of simulation with "gene selection" (adapted from Demo10). Each data is the mean of at least 200 simulations.

Sexual Selection:		High			None		
Polyploidy (Nr. alleles):		1	2	3	1	2	3
Type of selection process simulated							
Gene	Monosex	85	6	5	97	2	1
selection	Bisex	4	0	0	0	0	0
	Trisex	0	0	0	0	0	0
Group	Monosex	7	1	0	54	17	6
selection	Bisex	54	7	2	9	1	0
	Trisex	25	2	1	3	0	0
Isolated	Monosex	69	42	33	77	59	50
population	Bisex	98	83	70	91	61	48
survival	Trisex	98	75	38	90	43	24

Table 5-II: Mean variance of allelic distribution of selected genes in surviving isolated population after 20 time steps of the simulation (mean of 200 simulations).

Sexual Selection:		High			None		
Polyploidy (Nr. alleles):		1	2	3	1	2	3
Type of selection process simulated							
Neutral	Monosex	0.35	4.37	5.95	0.70	4.18	6.25
genes	Bisex	6.15	6.60	6.97	5.90	7.37	7.02
	Trisex	7.24	7.13	7.51	6.08	6.33	7.05
Genes for	Monosex	0.21	6.43	8.79	0.24	6.26	8.61
resistance	Bisex	0.07	0.82	1.81	0.22	2.59	4.86
	Trisex	0.09	1.50	3.90	0.34	3.12	6.39
Gene for	Monosex	0.03	1.06	2.69	0.06	1.14	3.29
clutch size	Bisex	0.46	1.77	2.31	0.52	1.51	2.69
	Trisex	1.12	3.28	4.08	0.90	3.03	4.28

6 The adaptive value of sex

Adapted from: Jaffe 2000

ABSTRACT: Further simulations with Biodynamica study the simultaneous effect of variable environments, mutation rates, ploidy, number of loci subject to evolution, and random and assortative mating, on various reproductive systems. Assortative mating is defined here as mating between genetically similar individuals. Novel simulations with "gene selection" showed that mutants for sex and recombination might become evolutionarily stable, displacing alleles for monosexuality in diploid populations mating assortatively under variable selection pressure. Assortative mating reduced excessive allelic variance induced by recombination and sex, especially among diploids. Results suggest a novel adaptive value for sex and recombination and show that the adaptive value of diploidy and that of the segregation of sexes is different to that of sex and recombination. The results suggest that the emergence of sex had to be preceded by the emergence of diploid monosexual organisms and provide a explanation for the emergence and maintenance of sex among diploids and for the scarcity of sex among haploid organisms.

The mysterious existence of sex

What selective forces maintain sexual reproduction and genetic recombination in nature? The answer to this question is an elusive mystery (Maynard-Smith 1978, Judson and Normak 1996, Hurst and Peck 1996). Even simulations of Biodynamica presented in the previous chapter, where sex emerged and was maintained under group selection scenarios, did not found the conditions under which sex could emerge and evolve under gene selection scenarios.

Asexual reproduction is theoretically much more likely to occur than sexual one due to at least three inherent advantages: parthenogenic females do not need to find mates; they produce twice as many daughters and four times as many granddaughters compared to the average sexual ones; and natural selection drives adaptation and thus selection of relevant genetic traits much faster in asexual organisms compared to sexual ones (Maynard-Smith 1978, Jaffe 1996). Despite these relative theoretical advantages of asexuality, most higher organisms are sexual. The various hypotheses put forward to explain this mystery can be grouped into three broad categories:

1- The ecological genetic models and the Red Queen Hypothesis which postulate that sex is adaptive in variable environments or variable parasite pressure because it enables genetic variation and the rapid spread and creation of advantageous traits (Bell and Maynard-Smith 1987, Hamilton et al 1990, Ebert and Hamilton 1996, Howard and Lively 1994).

2- The mutation-accumulation models (Muller 1964, Hill and Robertson 1966, Kondrashov 1984, 1988, 1994, Taylor and Williams 1982, Heisler 1984), which suggest that sex is adaptive because it performs the efficient removal of deleterious mutations or DNA repair.

3- The mate selection models, which assume that sex allows for the selection of 'good genes' by orientating the evolutionary process towards the fixation of beneficial traits (Kodric-Brown and Brown 1987**,** Jaffe 1996, 1999). Specifically, assortative mating has been shown to be very successful in increasing the fitness of sexual species (Davis 1995, Jaffe 1998). Non-random mate selection was shown to make sex advantageous in group selection scenarios (see above), but not under gene selection.

The role of mate selection in stabilizing sex (see also next chapter)

Regarding the mate selection models, computer simulations showed that non-random mate selection accelerates evolution by focusing natural selection on the appropriate genes (Jaffe 1996),

especially in the case of assortative mating (Davis 1995, Jaffe 1998). Mate selection may explain evolutionary phenomena such as sympatric speciation (Todd and Miller 1991), changing adaptive behavior (Miller and Todd 1994), and behavioral diversity (Werner and Todd 1997). Mate selection strategies aiming at reducing mutation loads were slightly better than random mating (Kondrashov 1988), and assortative mating improved the fitness of simulated populations up to four times compared to random mating (Jaffe 1988).

The concept of assortative mating used here includes passive and active assorting. Active assorting means that individuals chose similar mates by actively searching for them, using mechanisms such as imprinting of sex models (Lorenz 1935, Kendrick et al 1998) for example; whereas passive assorting means that mating may occur among genetically similar individuals due to population viscosity, low motility, inbreeding and/or geographical or ecological restrictions (Peck et al 1998, Queller 1992, Peck 1996), which make mating with neighbors more likely and at the same time make neighbors genetically more similar. In both cases the result is that mating occurs among genetically similar individuals.

Previous work (Jaffe 1999) showed that assortative mating might reduce excessive allelic variability caused by sexual reproduction, especially when selection acts on various loci simultaneously. Assortative mating also allows fit females to maintain (not diluting) their advantageous genetic composition during reproduction, providing their offspring with a similar advantageous genetic outfit. It may accelerate the extinction of sub-optimal genetic combinations, as contrary to strategies selecting for good genes, assortative mating induces suboptimal females to mate with suboptimal males, thus accelerating the extinction of suboptimal alleles. The intensity of mate selection affected the success of assortative mating, as excessive screening for potential mates diminished the efficiency of mate selection, slower so in large populations.

The role of ploidy

The ploidy of organisms is also relevant to the evolutionary dynamics of sex. Asexual ploidy cycles have been proposed as leading to the origin of sex by reducing mutation load (Kondrashov 1994), and poliploidy has been shown to maintain higher genetic variance under strong selection (Jaffe 1996). However, most studies on the emergence of sex have focused on haploid organisms (Hamilton et al 1990, Kondrashov 1988, Miller and Todd 1994, Peck et al 1998 for example). The genetic complexity (number of loci subject to adaptation) of the simulated organisms also affects the evolutionary advantage of sex and recombination (Maynard-Smith 1978, Jaffe 1998), as does the complexity of the changing environment simulated (Maynard-Smith 1978, Hamilton et al 1990).

The evolutionary dynamics of sex

Here I will present results from a study of the dynamic evolutionary interaction between ploidy types, mutation rates, assortative mating, sex determination mechanisms, variable numbers of loci subject to adaptation and changing environmental selection pressures, showing features which are not evidenced when studying these systems on their own, but which emerge as a result of the interaction.

The results of the various possible combinations of variables explored with over 120,000 simulations of gene selection are summarized in Figures 6-1 and 6-2. In these figures, data points greater than one indicate that the respective allele was more frequent than the corresponding monosexual variety. The most striking result is that alleles coding for sexuality displaced corresponding alleles coding for monosexual strategies when simulating hermaphrodites and diploid bisexual organisms which mated assortatively and produced more females than males. For example, simulated diploid hermaphrodites of type 1 (D-Herma1) organisms mating assortatively displaced diploid monosexual (D-Monosex) organisms which did not mate but reproduced asexually in a ratio >4/1 when simulations included 8 loci with allelic variance (Fig 6-1). Haploid organisms or diploids using random mating did not succeeded in displacing the corresponding monosexual variety.

The relative advantage of sexuality over monosexual reproduction was dependent on a variety of factors in a non-linear pattern. More than four loci with allelic variance in the simulated organisms were needed for sex alleles to displace monosexual ones (Fig 6-1), and low mutation rates favored sexuality (Fig. 6-2). As had been shown before (Maynard-Smith 1978, Peck et al 1997) small population sizes also favored sex (not shown).

Experiments in which all individuals were monosexual in the initial population and during the simulation sexual organisms were allowed to appear through random mutation, or vice versa (Table 6-1) showed that alleles coding for sexual diploid hermaphrodites with assortative mating are evolutionarily stable vis-a vis alleles coding for monosexual reproduction and are clearly able to invade and displace monosexual diploid alleles from the populations. Alleles coding for other diploid hermaphroditic sexual strategies, although to a lower degree, may also displace eventually monosexual alleles. Bisexuality seemed unable to displace monosexuality in this scenario.

The effect of allelic dominance

The effect of allelic dominance on the evolutionary dynamics was studied using the simulation of populations consisting of a mix of D-Herma1 and D-Monosex with 7 variable genes. The simulations using random allelic expression of the phenotype (See Figure 6-1, Assortative mating) gave a mean value of $3.87 \pm$ sd 1.06 more alleles coding for D-Herma1 than those coding for D-Monosex under the conditions described in Figure 6-1. When simulating the same situation but including allelic dominance, so that the alleles coding for D-Herma1 were dominant and those coding for D-Monosex were recessive, the relative advantage for D-Herma1 alleles increased (mean ratio = 5.43 ± 1.36). When simulating dominance of D-Monosex so that D-Herma1 alleles were recessive, the evolutionary advantage of D-Herma1 alleles remained, although to a lower degree (mean ratio = 2.48 ± 0.29). Repeating experiments in Table 6-1 using dominant alleles for sex (D-Herma1-AM) increased the likelihood for successful displacement of D-Monosex by D-Herma1-AM from 52 % to 71 %, whereas when sex alleles were modeled as recessive, this likelihood decreased to 30 %. That is, although simulation of dominance affected the results, the evolutionary advantage of sex was maintained even if genes coding for sex were recessive.

The importance of assortative mating in regulating genetic variability

It has to be noted that assortative mating here, although modeled having mate selection in mind, can be extrapolated to gamete selection. For example, most sexual marine invertebrates simply spawn their gametes. The corresponding metaphor in the model would be that female gametes assortatively select male gametes through biochemical mechanisms after contact, or through passive assorting, by the fact that the probability of successful encounters between gametes and of subsequent fertilization is inversely proportional to the distance between the emitters of the gametes. A similar case can be made for sperm selection in which females not only may select males but also chose from different spermatozoa from one or several males (see Haig and Bergstrom 1995 for example). In the light of the findings here described, it would seem worthwhile to simulate sperm production in large amounts explicitly, modeling each spermatozoon with a slightly different allelic load.

The results suggest that the genetic variance produced by sex differs from that produced by random mutations in that sex with assortative mating produces a better blend of variation, allowing faster adaptation in scenarios with very large genetic combinatorial possibilities, if compared to random mating. That is, sex slows the speed of evolution (Jaffe 1996) as advantageous mutations are not always transmitted to the offspring and are often mixed with disadvantageous alleles in other loci during recombination. Asortative mating reduces the extent to which this "dilution effect" of advantageous mutations occurs (Jaffe 1999), by reducing the variance of allelic composition between mates and thus producing offspring that have a greater likelihood of possessing the advantageous genes of their parents. Thus, assortative mating accelerates the fixation of advantageous alleles in the population canceling the effect of sex in slowing evolution. On the other hand, the long-term advantage of sex is that it can

produce advantageous blends of alleles faster than asexual reproduction does, but only if the number of loci is large (Jaffe 1998). For genomes with low genetic complexity (number of loci), mutations together with asexual reproduction is faster than sex in achieving optimal allelic combinations in the genome. Thus, the advantage of sex will be evidenced only if organisms do not mate randomly and the simulated genome has sufficient complexity (Ochoa and Jaffe 1999). Simulating diploidy here is in fact a way of increasing genetic complexity, making the advantages of sex much more evident. Our results (Fig. 6-1) though show that the complexity produced by diploidy is not identical to the complexity produced by an equivalent increase in the number of loci, due to the particularities of meiosis. An approximate representation of the dynamics of these processes is given in Figure 6-3.

Why random mating is unlikely to occur

Most studies on the emergence and maintenance of sex have focused on models using random mating. The present results suggest that the emergence of sex seems to be favored when the following conditions are present simultaneously: Variable environments (i.e Red Queen Hypothesis), assortative mating, low mutation rates, small founder populations, diploidy, hermaphroditic sex, and many loci suffering adaptation. These assumptions are compatible with what seems to happen with founder populations in biological allopatric speciation (Mayer 1988). That is, in founder populations variable environments select alleles in several different loci at the same time and assortative mating, i.e., mating among individuals in the founder population, is very likely to occur. Thus, sex, if not present, may emerge with higher probabilities when new species evolve, compared to a scenario were sex emerges in an established species.

Complexity, assortative mating and sex

The new features analyzed here, not included in the exhaustive review on the emergence and maintenance of sex by Maynard-Smith (1978), are assortative mating and genetic complexity. The most likely scenario for the emergence of sex, in the light of these new findings, is that sex may have emerged from diploid monosexual ancestors, and that mating occurred preferentially among individuals with the same predisposition for sex, creating the simplest form of assortative mating. This means that the evolutionary step from haploidy to diploidy had to precede the emergence of sex, so that organisms evolved from asexual haploids, to haploids which occasionally interchanged genetic information, to monosexual diploids, to bisexual diploids. Sexual haploids, thus, from this point of view, have to derive from sexual diploids. It has been proposed that anisogamy also stabilizes sex (Kodric-Brown and Brown 1987). The present simulations support this suggestion but they also showed that anisogamy is unlikely to displace monosexual reproductive strategies in one single evolutionary step. The body of evidence now available suggests that the adaptive value of diploidy, of recombination in monosexual diploids, of the exchange between organisms of genetic material (i.e. sex), and of anisogamy differ, as each process has a different effect on the evolutionary dynamics.

Assortative mating favors the maintenance of sex and recombination even among bisexuals, overcoming the two-fold disadvantage of sex relative to parthenogenesis. Other mate selection strategies, not explored here (but see Jaffe 1999), may produce similar biases towards sex, as is the case of mate selection criteria looking for "good genes" (Jaffe 1996, 1998). A testable prediction, derived from the simulations, is that sex should be rare or absent among haploid organisms, and that sex is maintained by selective forces which are different from those favoring diploidy (Kondrashov 1994) or polyploidy (Maynard-Smith 1978, Jaffe 1996) .

Experimental parameters used

Simulations were "tournaments" between two alleles coding for reproductive strategy (gene 7, Table 4-1), in interbreeding populations. When creating the initial population, each individual was outfitted with only one alleles at the gene coding for reproductive strategy, and with a set of alleles chosen at random (uniformly distributed) for each of the remaining genes. All organisms had the same

allele for gene 0 in each simulation. The temporal variations of the relative frequency in the population of the two alleles coding for reproductive strategy was monitored. The tournament tested which allele for reproductive strategy was able to displace the other. Results represent the average frequency of a given allele after a given number of time steps, calculated from hundreds of simulations having the same parameters but initiated with different random seeds. The random seeds were also chosen at random.

The reproductive strategy could be for haploid (**H**) or diploid (**D**) organisms. If sexual (i.e. not monosexual), organisms could mate randomly (**RM**) or assortatively (**AM**). Thus, ten different reproductive strategies were simulated. Monosexuals simulated parthenogenesis or thelytoky. That is, monosexual organisms did not mate. In **H-Monosex** (monosexual haploids), the individual transmitted all its genes to the offspring (cloning) with no variance except that allowed by mutations, simulating asexuality. **D-Monosex** (monosexual diploids) did not mate and produced offspring by uniform random crossovers of the alleles in each loci of the parent. Bisexuals (either H- or D- and -RM or -AM) produced equal numbers of males and females randomly (**Bisexual-r**) or produced a biased ratio of 60 % more females (**Bisexual-b**). Males could mate several times each reproductive step. Hermaphrodites (either H- or D- and -RM or -AM) produced only females and reproduced similar to bisexuals if finding another hermaphroditic female (**Herma1**) or any female (**Herma2**), or else reproduced as the corresponding H- or D- monosexuals. Herma1-RM, thus, mated assortatively with females having the same disposition for sex, even when mating randomly regarding all other loci.

Females produced offspring according to their phenotypically determined clutch size, transmitting their genes following Mendelian rules of reproduction (free recombination). If sexual, each parent provided half of its alleles to the newborn, so that for each locus, one allele came from each parent if diploid, or each parent had a probability of 0.5 to transmit its allele to each locus if haploid.

Optimal size of populations was 400 and the initial size of the populations was 200 individuals.

Simulations of genes with allelic variance allowed mutant alleles to appear in the range given below. Initial populations had individuals possessing any of the alleles indicated in that range. In simulations in which some genes had no allelic variance, the default allele, indicated in parenthesis, was assigned to all the corresponding loci in all organisms. The genes used here were:

Gene	Range for alleles	Effect on phenotype
8	1-2	Ploidy. Either haploid or diploid.
7	1-6	Reproductive strategy
9	0-10	Mutation rate: from 0.2 to 10^{-7} mutations per gene in logarithmic decrements (0.008)
2	0-10	Maximum life span coding for life spans from 0 to 10 time steps (5)
3	0-10	Clutch size from 0 to 10 offspring (5)
4	0-5	Minimum age for initiating reproduction of females in t-steps (0)
10	0-10	Resistance to biocide 1: Only allele 0 was resistant to that biocide (0)
11	0-10	Resistance to biocide 2: Idem as gene 6 but for biocide 2 (0)
12	0-10	Resistance to biocide 3: Idem as gene 6 but for biocide 3 (0)

Relevant Demos of Biodynamica: Demo17 to Demo19

Table 6-1: Percentage of simulations of "gene selection" where the strategy that appeared through mutation (invading strategy) displaced eventually all individuals possessing alleles of the original strategy (possessed by all individuals initially) in less than 200 time steps (250 simulations for each pair). A mutant of the strategy not present in the initial population was allowed to appear at random after time step 5. Other parameters were as in Fig 6-1 with 7 loci (see Demo19).

Invading strategy vs. original strategy: Percent of successful displacement:

When original population was homogeneously monosexual
D-Herma1-AM vs D-Monosex   52 %
D-Herma1-RM vs D-Monosex   31 %
H-Herma1-RM vs H-Monosex   9 %,
H-Herma1-AM vs H-Monosex   6 %
D-Bisex-b-AM vs D-Monosex   1 %
When original population was homogeneously sexual
D-Monosex vs D-Herma1-AM   7 %
D-Monosex vs D-Herma1-RM   23 %
H-Monosex vs H-Herma1-RM   17 %,
H-Monosex vs H-Herma1-AM   22 %
D-Monosex vs D-Bisex-b-AM   35 %

Figure 6-1: Frequency of alleles coding for sex relative to those coding for the corresponding monosexual population for varying genetic complexity (number of loci having allelic variance) as given in the x-axis (in the order j = 8, 7, 9, 2, 3, 4, 10, 11, 12 of Table 4-1). Each data point is the average value at time step 20 (which in average represented 10 generations of agent-organisms) of a minimum of 200 simulations and until standard deviation was < 30 % of the mean. Relative frequency < 1 indicate prevalence of monosexual allele. Data for 1 locus represents evolution with no selection other than caused by the dynamics of the reproductive strategy. Results for H-Herma1 mating randomly seem analogous to those reported in (Hamilton et al. 1990). (Adapted from Demo17 and Demo18)
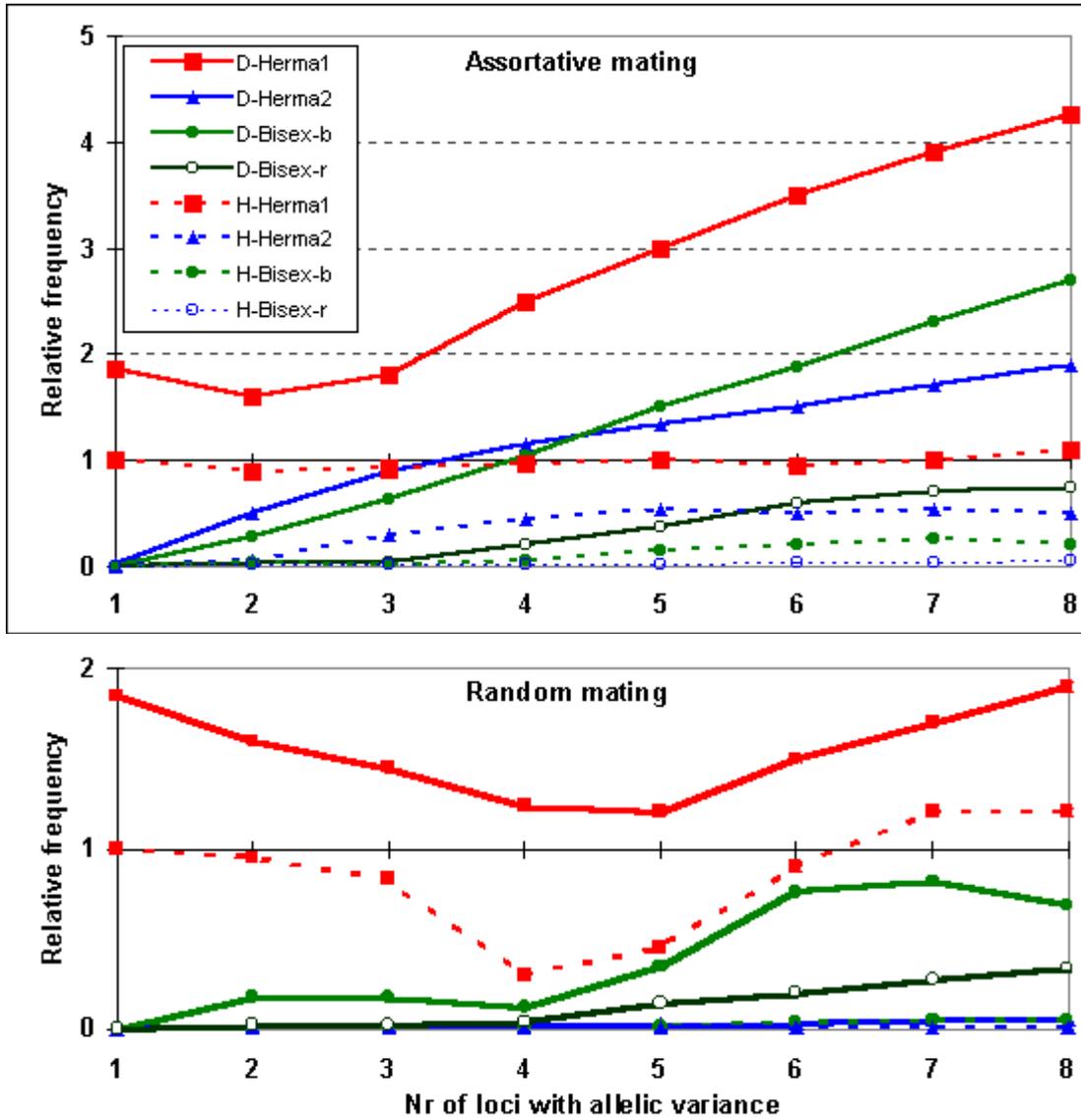
Figure 6-2: Relative frequency at the end of simulations of alleles coding for sex relative to those coding for monosexuality at different mutation rates. The loci j = 8, 7, 2, 3, 4, 10 of Table 4-1 had allelic variance. Mutation rates are given in random mutations per locus per generation. Else as in Fig 6-1.
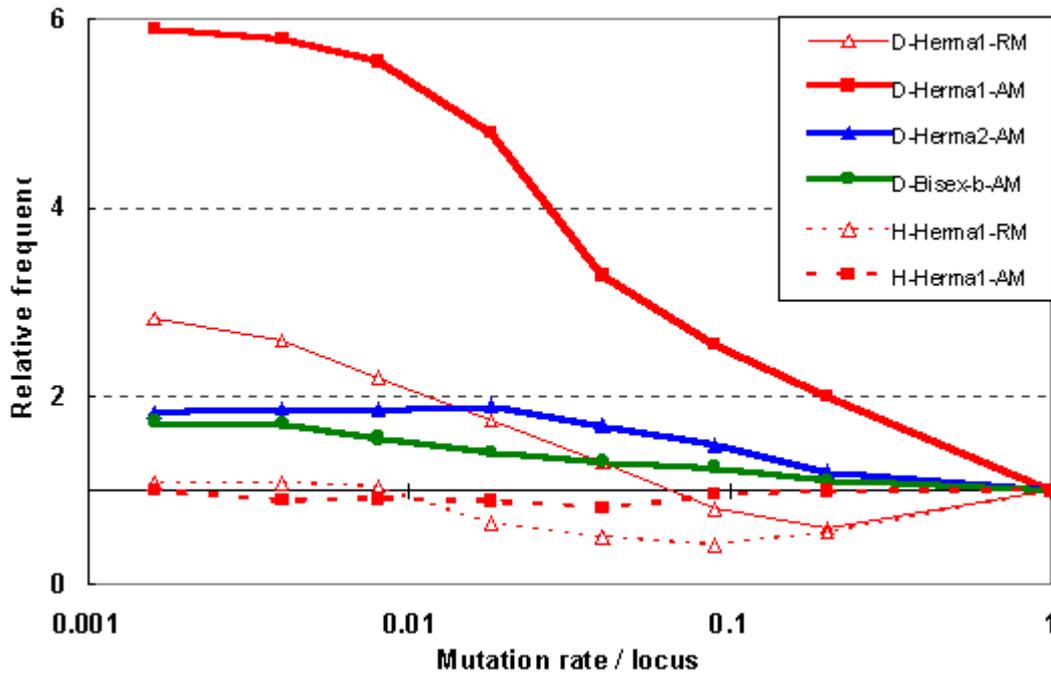
Figure 6-3: Approximate time course of mean fitness of organisms in an evolving population using different reproductive strategies.
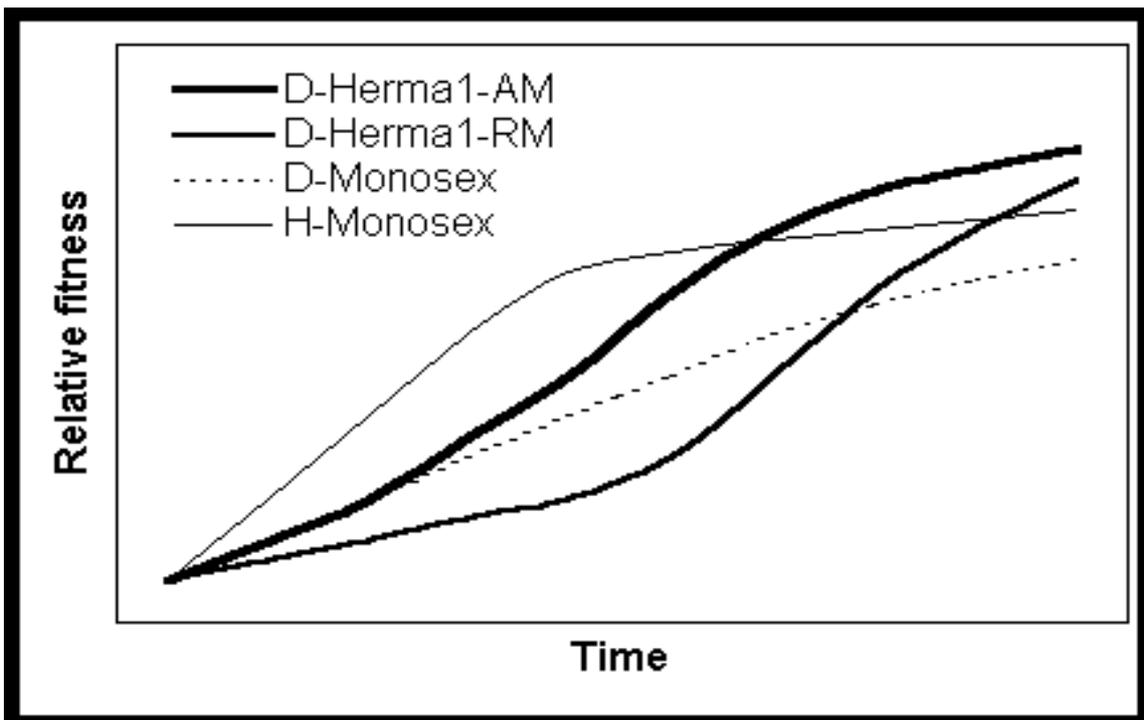
Experimental Evidences

One of the predictions of the simulation results is that sex is not always adaptive. Thus non-sexual strategies should be adaptive. Experimental evidence for this point can be found in the study of the sex life of nematodes:

BACTERIA DETERMINE THE DEGREE OF AMPHIMIX OF THEIR SYMBIOTIC ENTOMOPATHOGENIC NEMATODES

Taken from: Rincones, Mauléon and Jaffe 2001

Summary: Entomopatogenic nematodes show two different strategies in regulating the degree of amphimix as a response to nutritional stress. One strategy increases the production of males, increasing amphimix, increasing the genetic variability of the offspring, and thus improving the chances for a successful new adaptation. Another strategy increases the production of hermaphrodites at the expense of males, increasing the total number of reproductive individuals and thus the total number of offspring produced, increasing the odds that a few may survive to infest new insects. The strategy used depends upon the strain of symbiotic bacteria the nematodes are growing. The results help in discriminating between rival theories for the existence of sex.

INTRODUCTION: Although sex is ubiquitous, there is no generally accepted explanation for its existence. Do sex helps organisms to adapt to new or to changing environments by increasing genetic variance? Or, do organisms fare better under environmental stress when they are able to boost their reproductive output by switching to asexual reproduction? A recent theoretical model suggests that sex may be advantageous in solving evolutionary problems in highly competitive situations, if adaptation can proceed at a moderate pace and if the environmental changes force adaptive changes in several different genes simultaneously (Jaffe 1996, 2000). The model predicts that under environmental stress, a reversion from sex to no sex (or from bisexual to monosexual reproduction) is advantageous if the adaptation requires the involvement of a few (<4 genes). The reverse is true if the adaptation to the new environmental demands require the simultaneous change in various genes or loci. Here we want to explore the reproductive strategies used by facultative sexual nematodes when submitted to environmental stress. The nematodes in the family Heterorhabditidae are insect parasites that cultivate and feed on symbiotic bacteria of the genus *Photorhabdus*. The bacteria kept and transported by the nematode feed on dead insects (Kaya & Gaugler 1993). The life cycle of these entomopathogenic nematodes consists of eggs, four juvenile stages (instars) and adults. Third instars may become infective agents when, covered with the cuticle of the second instars, they stop feeding and leave the dead insect taking some bacteria with them in the intestinal tract to infect new insects. When infecting a live insect, the nematode enters the hemolymph, releases its symbiotic bacteria and substances that kill the insect in about 24-72 hours. Once inside an insect the nematodes reproduce either amphimictic (producing males and females) or by producing hermaphrodites which reproduce either automictic (auto fecundation), amphimictic or through matricidal endotoky (Strauch et al. 1995). In this endotoky, eggs are retained in the body of the mother and the juveniles grow inside their mother killing her and then forming infective juveniles. When nematodes are starved and/or nematode population densities are high, more infective juveniles are formed (Wang & Bedding 1996). This phenomenon may be regulated by pheromones (Golden & Riddle, 1984). The relationship between the nematode and the symbiotic bacteria seems to be species and strain specific (Wilkinson & Hay, 1997). These nematodes are known to produce variable proportions of males under different circumstances (Strauch et al. 1995). Here we explore the strategies used by these nematodes in regulating amphimix as a response to nutritional stress, as a test case for the proposed theoretical model.

MATERIALS AND METHODS: Nematodes of the species *Heterorhabditis bacteriophora* (Hb) (Poinar, 1976) and *H. indica* (Hi)(Poinar, Karunakab & David, 1992)were used. Samples of Hb were Hb1: FRG01 (Basse Terre, Guadeloupe, French Antilles), Hb2: HP88adams and Hb3: HP88 (both from Ohio, USA); and samples of Hi were Hi1: FRG20 (Guadeloupe) and Hi2: FRG22 (St. Barthelemy), all provided by the Laboratoire de Nematodes Parasits des Insects, INRA, Guadeloupe. Nematodes were reared on *Galeria mellonella* as described in (Woodring & Kaya, 1988). The bacteria were isolated using the "hanging drop" method described in (Poinar & Thomas, 1966) and reared on Petri dishes at 26-28°C over McConkey agar. Bacteria were named using the code for the nematode strain they originated from, preceded by the letter "B". Tests for presence of the correct bacteria included assessing the presence of catalaze using hydrogen peroxide and assessing the presence of antibiotics by testing production of toxins against *Microccocus luteus.*

The bacteria were grown on Petri dishes for 48 h and then a fixed number of nematodes (seed: either 50, 100 or 200 nematodes producing initial densities of 5, 10 and 20 infective juveniles per cm^2, see Zioni et al., 1992; Koltai et al., 1995) were placed in the dish. The dish contained for aga1 and agar2 respectively 16 and 8 g/l of meat extract nutrient broth, 11 and 0 ml/l wheat oil and 15 g/l agar-agar. After 6 days (when second generation adults matured), nematodes were killed and fixed in TAF (2 % triethanol-amine + 3 % Formaline) and individuals were counted.

RESULTS: Nematode populations cultured on bacteria growing on the poorer agar2 where always smaller than those cultured on richer agar1 ($p < 0.0001$, t-test). The percentage of males among the adult population changed between agar2 and agar1, depending on the bacteria present in the culture (Figure 1A). That is, nematodes Hb2 and Hi2 cultured on their symbiotic bacteria (BHb2 and BHi2 respectively) produced a lower percentage of males in poor agar compared to richer agar, whereas nematodes Hb1 cultured on their symbiotic bacteria produced higher percentages of males in the poorer agar. Nematodes Hb3 cultured on bacteria BHb3 produced only juveniles and hermaphrodites while the same nematodes cultured on BHb2 and BHb1 produced also males (not shown). Nematodes Hi1 and Hi2 cultured on BHi1 did not grow under the experimental conditions used (not shown). When we exchanged the bacteria between the nematode strains, a pattern, practically identical to that shown in Fig 1A was observed (Fig. 1B). Thus, the pattern of male production among nematodes depended on the bacteria on which they were feeding rather than on the nematode strain. The exceptions were BHb2/Hb1-seed 100 and BHb1/Hb2-seed 200, where the nematode strain seemed to be a better predictor for the relative pattern of male production on the two types of agars. Extreme examples were Bacteria BHb3, which always made the nematode strain growing on them produce only hermaphrodites. The number of nematodes used to initiate the experiment (seed) had no important effect on the pattern of male production ($p>0.1$, MANOVA).

DISCUSSION: Results showed that at least two different strategies for male production under dietary stress are used by these nematodes. One strategy increases the proportion of males produced, whereas the other increases the proportion of hermaphrodites produced when nematodes suffer dietary stress. The type of strategy chosen is in great part determined by the symbiotic bacteria. Both strategies have adaptive advantages. An increased production of males increases the chances for amphimictic reproduction, eventually increasing genetic variability of the offspring, improving the chances for successful adaptation to dietary stress. An increased production of hermaphrodites (and thus lower production of males), increases the total number of reproductive individuals and thus the total number of offspring produced, increasing the chance that a few may survive to infest new insects. It is intriguing that the strain of symbiotic bacteria determines what strategy the nematode is to use, the more so as the specific adaptive advantage for the bacteria or for the nematode of each strategy are unknown. It is though tempting to speculate, based on the results of the theoretical model, that the mechanisms uncovered here optimize the speed of adaptation. That is, the bacteria induce the switch to sexual reproduction in nematodes whenever they are overwhelmed by environmental change, whereas asexual (or monosexual) reproduction is favored when bacteria can cope with the environmental change, as they themselves reproduce mainly monosexually.

Figure 1: Min-max, 25%-75% range and Median value for the percentage of males among the adult
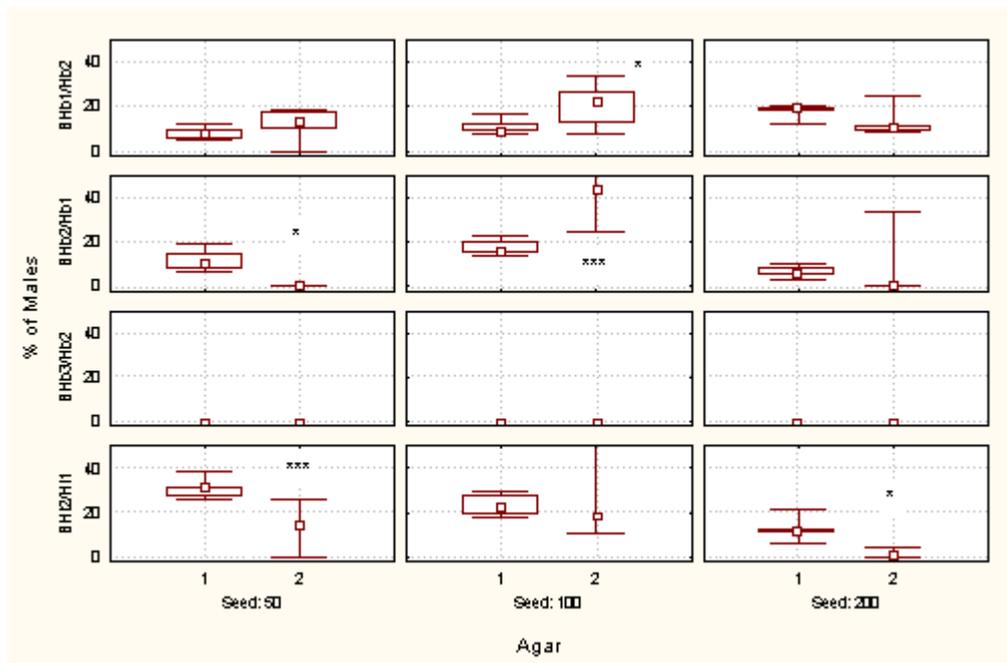
population in experiments with different bacteria/nematode combinations growing on two different agars (Agar1 has about twice as much nutrients as agar2). A: Experiments growing nematodes on their own bacteria. B: Experiments growing nematodes on bacterial
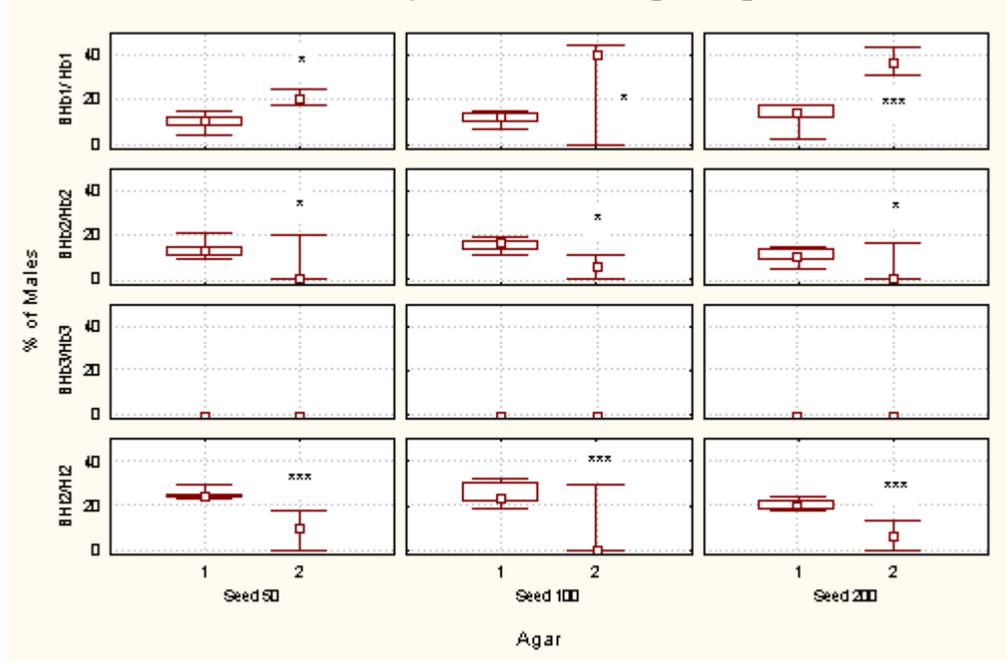
cultures from other nematode strains.

A:

B:

Data with * and *** differ from those of agar1 at p< 0.05 and p< 0.001 respectively using LSD on transformed data using formula y = arcsin(sqrt(x/100)). Breakdown Anova for the data gave values of F= 5.9, df = 23 x 71, p < 0.0001 and F= 6.3, df = 23 x 69, p < 0.0001 for data on graphs a and b respectively

7 On the adaptive value of love

Adapted from: Jaffe 1999

ABSTRACT: Results of simulation with Biodynamica of the evolution of diploid sexual organisms showed that several mate selection strategies confer much higher average fitness to the simulated populations, and higher evolutionary stability to the alleles coding for these strategies, than random mating. Strategies that select for 'good genes' were very successful, and so were strategies based on assortative mating. The results support the hypothesis that mating is not likely to be random in nature and that the most successful mate selection strategies are those based on assortative mating or on advantageous genes. The results suggest that sexual populations in nature should avoid both, maximizing outbreeding or maximizing inbreeding, and should acquire mate selection strategies that favor optimal ranges of genetic mixing in accordance to environmental challenges.

The importance of mate selection

Although there is little evidence for mate selection in species reproducing sexually, there is no evidence for the absence of mate selection in any sexual species. That is, sexual reproduction without previous courtship behavior (or gamete selection) may be rare, as even yeast have been suggested to have mate choice (Nahon et al. 1995). Mate selection or courtship is commonly thought to assure that mates are of the same species and of different sex. However, most organisms have courtship behaviors that seem too elaborate to serve only this purpose. Thus, Darwin coined the concept of sexual selection, which has acquired a variety of meanings (Andersson 1994, Andersson and Iwasa 1996, Holland and Rice 1998, Murphy 1998). In addition to courtship, sexual reproduction may be associated with sophisticated gamete selection mechanisms. For example, studies of female choice (Eberhard 1996) suggest that sperm selection, as a complement to mate selection, is a rather generalized characteristic of organisms.

The effect of mate selection on biological evolution may be very complex and is not easily characterized with analytical models. A few studies based on simulation models (Miller and Todd 1994, Davis 1995, Jaffe 1996, 1998), showed that mate selection affects biological evolution significantly. Theoretical studies of mate selection coupled to natural selection suggest that mate selection may reduce mutation load (Maynard Smith 1978, Taylor and Williams 1982, Heisler 1984, Kondrashov 1988), it may accelerate evolution by focusing natural selection to the appropriate genes (Jaffe 1996), it may be fundamental in explaining sympatric speciation (Todd and Miller 1991, and see references in Andersson 1994, and in Price 1998, for example), changing adaptive behavior (Miller and Todd 1994), and behavioral diversity (Werner and Todd 1997).

A problem in exploring the possible impact of mate selection on the evolutionary dynamics is the large amount of mate selection strategies possible. The strategy chosen will affect the results. Mate selection strategies aiming at reducing mutation loads showed a small improvement over random mating (Kondrashov, 1984, 1988), but assortative mating improved the fitness of population up to 4 times compared to those using random mating (Jaffe 1998). This improvement of assortative mating was evidenced only if the simulated organisms had more than 7 genes. Therefore, I used Biodynamica, simulating organisms with 14 genes, to explore the effect of various mate selection criteria on the evolutionary dynamics of sexual organisms, as this simulation model allows the study of features that emerge as a result of the interaction between mate selection and natural selection.

The adaptive value of mate selection strategies

The effect of each of these mate selection criteria on evolution in diploid organisms is shown in Figures 7-1 and 7-2. Figure 7-1 shows the results of the evolution experiments simulating isolated populations, each with a given mate selection criteria (MSC), whereas Figure 7-2 presents the results of simulations of gene selection. These results may be grouped into three categories:

1- Trivial but important results: All three MSC in which females chose males according to beneficial genetic traits (MSC 3, 4 and 5 in Fig 7-1) outperformed random mating when these strategies where tested in isolated populations. That is, the more good genes are selected through mate selection, the greater the average fitness of the population (Fig. 7-1) and the more probable that the alleles coding for these MSC are maintained in a population (Fig. 7-2). These results suggest that selection criteria that somehow increase the odds of mating with a fitter mate are very successful. Several examples of secondary sexual characters as fitness indicators in real organisms are known (Eberhard 1985, Andersson 1994, for example). The model predicts that the more genes are associated to a sexual signal, the more likely that it is selected for mate selection. This seems to be the case, for example, of bilateral symmetry as a cue for mate preference (Pennisi 1995), where several genes affect directly or indirectly the degree of symmetry of the organism, which in turn determines the odds to be selected by a mate.

2- Surprising results: Pure sexual selection (MSC 1 in Fig 7-1) in which females chose males with strong sexual signals or sex appeal not directly related to fitness, induced rapid fixation of the allele producing the highest sex appeal (in less than 15 time steps in 95 % of 200 simulations). Populations with this mate selection strategy had a slightly higher but statistically significant survival probability compared to random mating (Fig. 7-1, chi-squared test between MSC 0 and MSC 1: $p < 0.01$, 500 simulations). In addition, when gene selection was simulated, a slight but continuous increase over time in the frequency of alleles coding for this strategy was observed (Fig 7-2b). These results help validate the model as they show that sexual selection increases mating success of the emitter, and thus increase its fitness. The results also show that the simulations produce run-away sexual selection (Houde and Endler 1990, Bakker 1993, Houde 1994) in that stronger sexual signals (i.e. sex appeal) are fixed very fast by the evolutionary dynamics. The increase in the fitness of populations formed by individuals which had only the MSC 1 allele (Fig. 7-1) has to be explained due to genetic drift (Soler et al. 1996, Jaffe 1996), as the signal for sex appeal my frequently be associated to the presence of good genes (at least in these simulations), being then a good predictor of fitness. The simulation will then select organisms with both, good genes and the appropriate signal for sex appeal, even if the algorithm used in the simulation does not explicitly relate sex appeal with any additional trait conferring higher fitness. An indirect confirmation of these results is that recent evidence suggests that sexual signals that somehow increase mating success induce divergence of the trait mainly due to sexual selection (Arnqvist 1998), showing that sexual selection may fix genetic straits faster than natural selection.

The handicap simulation (MSC 2), consistently failed to improve on random mating (MSC 0). The adaptive disadvantage of the simulated handicap seemed to be too strong, and thus, populations in which females selected males based on this handicap had low survival probabilities (Fig 7-1), and alleles coding for this MSC were displaced by other alleles in interbreeding populations (Fig 7-2). This result could also be due to the fact that a high initial handicap does not allow the coupling of advantageous genes with the signal for handicap. The results suggest that sexual selection based on a handicap is likely only if the handicap is initially very small so that the signal may be coupled to good genes by the evolutionary dynamics (see also Andersson and Iwasa 1996, for example).

The fact that selection of old males (MSC 7) was less adaptive than that of young males (MSC 6), was unexpected (Fig. 7-1, $p < 0.005$, chi-squared test, $n = 200$). It might be thought that old males had shown they ability to survive and thus age should be an acceptable indicator for fitness. These simulations tell us otherwise. A tentative explanation here is that young males have recently been produced by the fittest parents and thus had a better genetic composition than older ones. The relative advantage of mating with old or young males may depend on the specific relation between live span and time steps used in the simulation and was not explored further.

3- Assortative mating: An interesting result was found with simulations of assortative mating, confirming its importance in biological evolution (Davis 1995, Jaffe 1998). Although active assortative mating was simulated, the results may also relate to passive assorting. That is, individuals may chose genetically similar reproductive mates actively or external constraints may force low dispersal rates, imposing mating among similar mates (high inbreeding). In both cases the result is that mating occurs among genetically similar individuals. Alleles coding for assortative msc (MSC 10 and 11 in Figure 7-1) dominated in simulations of gene selection (Fig 7-2), but when measuring the survival probability of isolated populations (Fig 7-1) it was assortative mating based on genes different to that coding for msc (MSC 8 and 11) that conferred relatively high survival probabilities to populations. Assortative mating based only on msc (MSC 10), in which females chose males with their same MSC, was particularly successful in simulations of gene selection (Fig. 7-2), but in simulations of isolated populations it was not better than random mating (Fig. 7-1, p > 0.1, chi-squared test, n = 200). That is, alleles for assortative msc had a strong penetrating power, displacing other alleles in simulations of gene selection, but did not improve survival probabilities relative to random mating. The advantage of assortative mating in simulations of gene selection is based on the fact that females select males which have their alleles for assortative mating, increasing the frequency of these alleles relative to other alleles, making it a strategy with high evolutionary stability in terms of the genetic dynamics, but with little effect on the average fitness of populations. Otherwise, assortative mating based on more general criteria (MSC 8 and MSC 11, Fig. 7-1), had a comparable fitness to MSC in which selection criteria were directly linked to advantageous genes (MSC 4 for example). Although it is not completely clear how assortative mating displaces other strategies in the simulations, several reasons (or a combination of them) may explain this: Assortative mating may reduce excessive allelic variability caused by sexual reproduction, especially among genetically complex organisms. It may allow fit females to maintain (not diluting) their advantageous genetic composition during reproduction, providing their offspring with a similar advantageous genetic outfit. It may accelerate the extinction of sub-optimal genetic combinations, as contrary to strategies selecting for good genes, assortative mating induces suboptimal females to mate with suboptimal males, thus accelerating the extinction of suboptimal alleles. The results showed that strategies based on assortative mating are the most likely to be selected through the evolutionary dynamics (i.e. gene selection, Fig 7-2), and thus should be common among real diploid organisms. Few cases have been thoroughly explored, although some controversial evidence for assortative mating among humans exist (Thiessen and Gregg 1980, Buss 1989, Rushton 1989, Grammer 1989, Voland and Engel 1990, Jaffe and Chacon 1995, but see the open peer commentaries in Rushton 1989 for example).

Selection by females of genetically different males increased allelic variability too much, making this strategy evolutionary unstable compared to random mating. The fact that dissortative mating was evolutionary unsuccessful is contrary to some findings in nature. For example, some organisms select their mates so as to increase the variability of the immune systems (Weedekind et al. 1995), by preferring mates with a different Major Histocompatibility Complex. This contradiction may be explained by the fact that in the present model, optimal allelic configurations existed. In simulations of the Red Queen hypothesis, in which co-evolution between parasites and host is simulated, dissortative mating based on genes conferring resistance to parasites showed to be more successful than assortative mating (Ochoa and Jaffe 1997).

The intensity of mate selection affected the success of the mate selection strategy. In the case of assortative mating (Fig 7-3), the screening of a maximum of five mates was sufficient to show a significant advantage over random mating. Excessive screening did not seem to improve the efficiency of mate selection, but rather tended to diminish it, although slower so in large populations.

Evolutionary impact of mate selection

The main result of the simulations is that mate selection is not likely to be random in nature. Several mate selection strategies (including strategies not explored here) may confer higher fitness and have a higher evolutionary stability than random mating. Thus, real organisms are very likely to use one or more mate selection criteria instead of mating randomly. Mate selection criteria, in order to increase adaptation, have

to screen a large number of genes. Thus, phenotypes, affected by a large number of genes are predicted to be very efficient as mate selection signals, as seems to be the case for bilateral symmetry (Moeller and Thornhill 1998). The present work predicts that a certain degree of assortative mating should be favored by evolution, and thus, should be common among real organisms. Clearly, data on mate selection criteria from plant and animal studies are insufficient to reach a strong conclusion on the subject, but this theoretical exercise may stimulate investigations in this direction.

The simulation model did not consider gamete selection (i.e. sperm competition and sperm selection, etc). Several of the mate selection strategies outlined here my work at the level of the interaction between gametes. Therefore, any practical prediction derived from this work should assess the outcome of the reproductive process, i.e. should study also the offspring rather than focus on mating alone. Two specific prediction derived from this work, which might be tested experimentally, are:

1- Assortative mating should be common in diploid and polyploid plants and animals. For example, pollination strategies that maximize outbreeding should be rare and where they exist plants should have poor seed dispersal mechanisms, making it likely that pollination occurs between neighboring plants that are genetically related.

2- Mate selection mechanisms in plant and animals are expected to be quite sophisticated in that individuals should select mates based on several criteria that reflect good genes and/or similar genetic composition. Excessive genetic similarity between mates is also to be avoided, so that for each species and population an optimal degree of genetic similarity between successful mates (i.e. gametes producing the offspring) should be expected.

The limits of assortative mating

One explanatory theory proposing a solution to the mystery of the existence of sex is called the **'Red Queen' hypothesis**, which states that sex is an adaptation to escape from parasites. In a paper by Ochoa and Jaffe (1999) this hypothesis was tested against the assumption that mate selection strategies confer advantages to sex, using an model similar to Biodynamica but which was developed independently. While previous simpler models testing the 'Red Queen' hypothesis considered mainly haploid hosts, stable population density, random mating and simplified expressions of fitness, this new more realistic model allowed diploidy, mate selection, live history constraints and variable population densities. Results suggest that the Red Queen hypothesis is not valid for more realistic evolutionary scenarios and that each of the two hypotheses tested seem to explain partially but not exhaustively the adaptive value of sex. The results of the simulations in Ochoa and Jaffe (1999) are consistent with simulations using Biodynamica, confirming the importance of mate selection as an evolutionary catalyst, favoring the maintenance of sex. Ochoa and Jaffe (1999) showed that selection by females of genetically different males increase allelic variability. For example, some organisms select their mates so as to increase the variability of the immune systems (Weedekind et al. 1995), by preferring mates with a different Mayor Histocompatibility Complex. In simulations where large genetic variability is not so important, assortative mating showed to be the most successful strategy (Jaffe 1999). These results, thus, allow for a falsifiable prediction. Organisms in nature should select their mates so as to achieve an optimal degree of allelic variability in their offspring. Excess genetic variability is not optimal (see above), nor is pure assortative mating. Thus, organisms should not maximize outbreeding or inbreeding but should strive for intermediate levels of genetic mixing. Studies of mate choice related to the genetic distance of the breeding pair in natural populations could provide data to settle this point. Although controversial, some indirect evidence for this is available for humans (Thiessen and Gregg 1980, Rushton 1989, Jaffe and Chacon 1995, but see the open peer commentaries in Rushton 1989 for example). Such evidence for plant pollination for example would be a very strong support for our hypothesis, as no data on mate selection in plants could be found in the literature. Another way of looking at this hypothesis is that mating in sexual organisms should aim at achieving optimal genetic mixes (not maximal mixes),. The optimal genetic mix should vary in accordance to environmental pressures. That is, populations (or species) colonizing new ecosystems for example should aim at larger genetic mixing compared to

populations adapted to stable niches, and thus, both group of populations should differ in their mate selection criteria. An alternative way to achieve improved genetic mixing in anisogamous species (Kodric-Brown and Brown 1987) when environmental challenges call for it would be an increased production of males or of sperm.

Experimental support for the existence of love in nature

All the simulations presented so far suggest that mating should not be random in nature. Mates should be chosen based on their genetic quality and/or on they genetic relatedness. That is, the best mate is a genetically fit individual that shares most, but not all, the alleles with the individual wanting to reproduce sexually.

Genetic quality can be assessed through phenotypes in multiple ways: Physical strength reflecting genetic qualities related to food retrieval, consumption and processing; symmetry reflecting genes producing harmonious enzyme assemblages allowing for perfect embryonic development; intelligence or other cognitive abilities reflecting a good working brain; precise pheromone blends for sexual attraction; etc. To my knowledge, all courtship behaviors known so far have features that allow for the assessment of one or more of these features. Some mate selection signals even, depend on environmental factors, as is the case in predatory mites that show diet-dependent female choice for males with 'good genes' (Lesna and Sabelis 1999). Even among plants, female flowers may select pollen based on a variety of physical and biochemical properties.

Genetic relatedness can be assessed in a multitude of ways. Great tit birds (*Parus major*), for example, choose they mates by relating the songs experiences by female songbirds early in life with those produced by their potential mates. Females tend to mate with males singing slightly unfamiliar songs, where familiar songs are defined as those sung by the female's father (McGregor and Krebs 1982). Another example are plants. They do not seem to maximize outcrossing but rather seem to prefer intermediate degrees, evidencing that optimal levels rather than maximal levels of autcrossing are preferred by most plants (Waser and Price 1983). More such examples are sure to appear in the literature.

Experimental parameters

The genes and their possible alleles defining the organisms simulated used are given below. Organism had allelic variance in 14 genes, except when simulating isolated populations where all organisms had the same allele for msc.

Gene Alleles Effect on phenotype

2	0-10	Maximum life span coding for life spans from 0 to 10 time steps.
3	0-10	Maximal clutch size.
4	0-5	Minimum age for initiating reproduction of females in t-steps.
6	1-2	Sex, either male or female.
13	0-10	Sex appeal of males towards females in an increasing range of attractiveness.
11	0-10	Resistance to biocide 2. Only allele 0 was resistant to that particular biocide.
12	0-10	Resistance to biocide 3 as for gene 6.
18	0-11	MSC or criteria for mate selection (Table 2).
15	0-5	Minimum age for initiating reproduction of males.
9	0-10	Mutation probability coding for mutation rates from 0.2 to 10^7 random mutations per gene with logarithmic increments.
14	0-1	Sex determination mechanism, either random or weighted according to value of gene 5.

5   0-10   Sex ratio of newborns.

19   1-100   Number of males screened by females in search of potential mates before mating.

1   0-10   Neutral genes whose phenotypic expression has no effect on fitness.

        The algorithms coded by the alleles of the gene responsible for the mate selection criteria (MSC) used by females in selecting conspecific males were (numeric nomenclature as in the figures, which differ from that in Table 4-1):

MSC   Mate selection strategy coded

0   Random mating: females mate with the first conspecific male encountered.
1   Neutral sexual selection: Females prefer males showing alleles with high values in their gene for sex appeal (gene 5 in Table I).
2   Handicap: Females prefer males with high values in their resistance gene 6, i.e. they prefer males susceptible to the biocide, which if alive is because they might (or might not) be better in other aspects which compensate for this susceptibility.
3   1 Good gene: Females prefer males with low values in gene 6, i.e., preferred resistant males, i.e., selection of one 'good gene'.
4   2 Good genes: Females prefer resistant males for both biocides, i.e., selection of two 'good genes'.
5   5 Good genes: Females prefer males with appropriate allelic values in genes for maximum life span, clutch size, age for females to start reproducing, and the two resistance genes (genes 1,2,3,6 and 7), i.e., selection of 5 'good genes'.
6   Youth: Females prefer young males.
7   Experience: Females prefer old males.
8   Assortative mating - msc: Females screen the allelic values of males and chose those with an allelic composition most similar to hers. All genes are screened, except gene 8 coding for MSC.
9   Dissortative mating: As MSC 8 but females prefer males with the most dissimilar alleles.
10   Assortative msc: Females prefer males that have her allele for *MSC*.
11   Assortative mating: Females prefer males with a similar allelic composition, screening all genes, including the gene for MSC (MSC 11 = MSC 10 + MSC 8).

        See Demo22 of Biodynamica

Figure 7-1: Probability of survival of 'isolated populations' in which all individuals are diploid and use an single mate selection criterion. Probabilities are expressed as a multiple of the probability of survival of populations using random mating. Mate selection criteria used are indicated with numbers in the ordinate, and correspond to the MSC as follows:

0: Random mating, 1: Neutral sexual selection, 2: Handicap, 3: 1 Good gene, 4: 2 Good genes, 5: 5 Good genes, 6: Females prefer young males, 7: Females prefer old males, 8: Assortative mating - msc, 9: Dissortative mating, 10: Assortative msc, 11: Assortative mating + msc

For each MSC, a minimum of 200 simulations were run for 30 time steps. Gene 8 (MSC) had a fixed allelic value in each simulation, as indicated in the ordinate, corresponding to the alleles defined above.
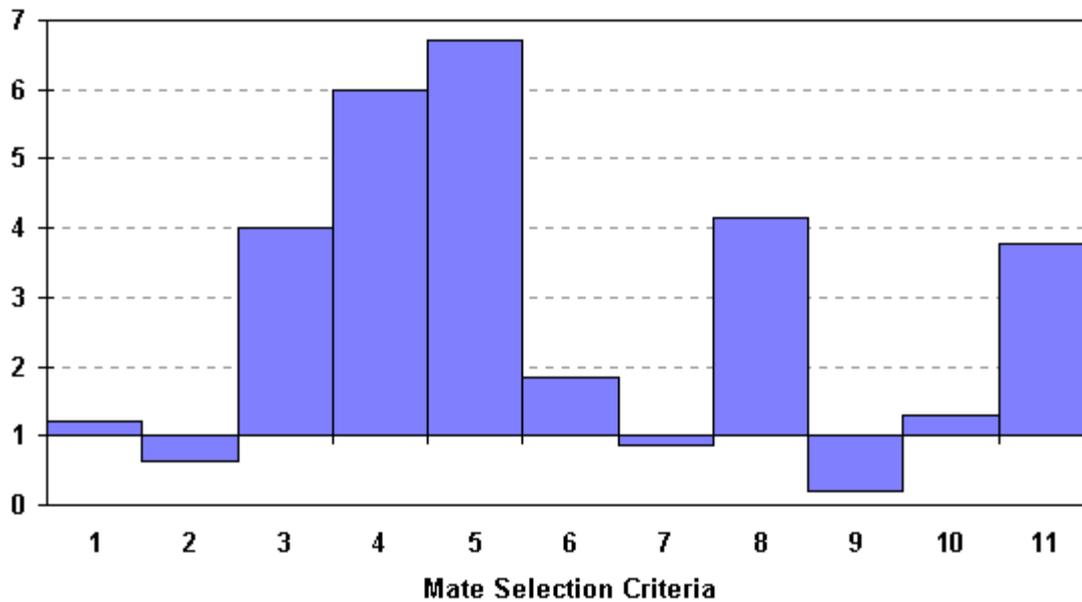
Figure 7-2: Average number of alleles in simulations of 'Gene selection' in which individuals coded their mate selection strategy according to alleles of gene 8 which has allelic variance. The y-axis gives the average among simulations of the absolute number of each allele present in the population at different time steps. Numbers indicated in the legend correspond to allelic values as in Figure 7-1. Else as in Figure 7-1. Fig. 7-2a includes strategies with assortative mating whereas Fig. 7-2b excludes them.

a
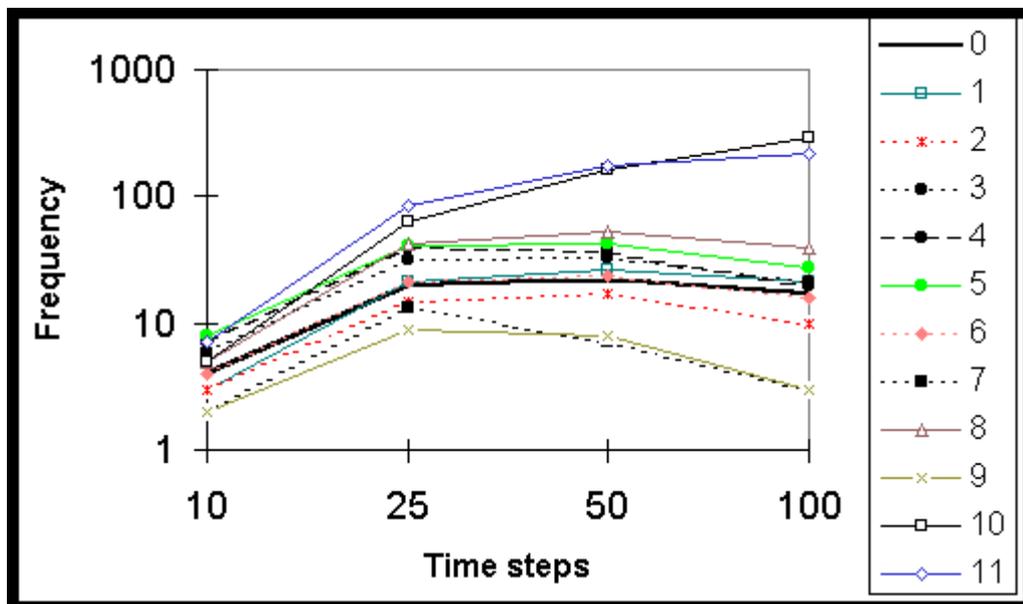
b
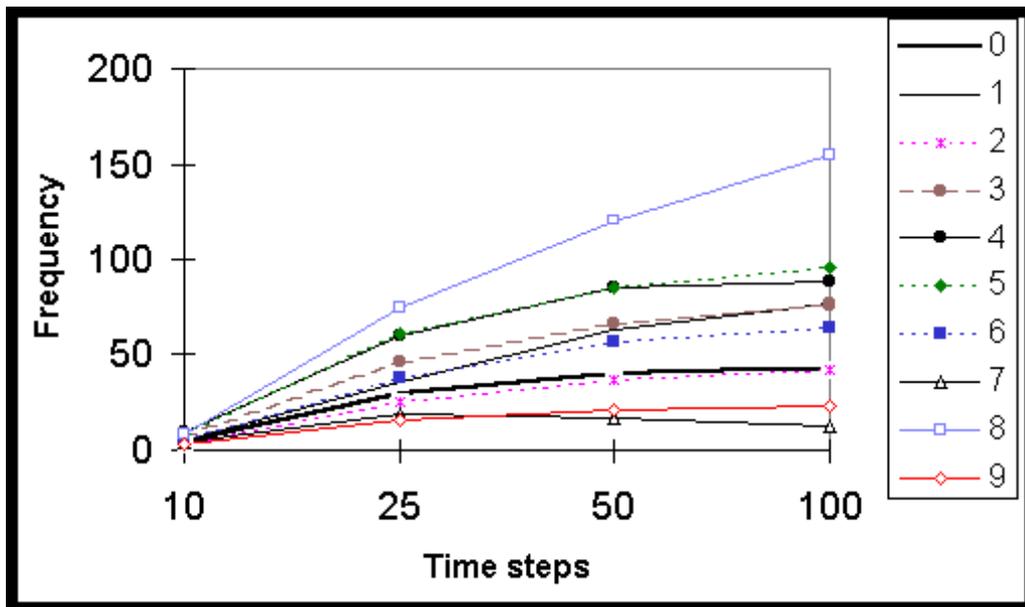
Figure 7-3: Effect of the mate selection intensity (number of males screened before mating) on the probability of survival of isolated populations of organisms using assortative mating. Values on the right y-axis indicated survival probabilities when the number of males screened was variable and was defined by alleles in gene 13, also subjected to adaptation. Curves indicated with squares, circles and triangles are for simulations with optimal size of the population values of 200, 400 and 800 individuals respectively.
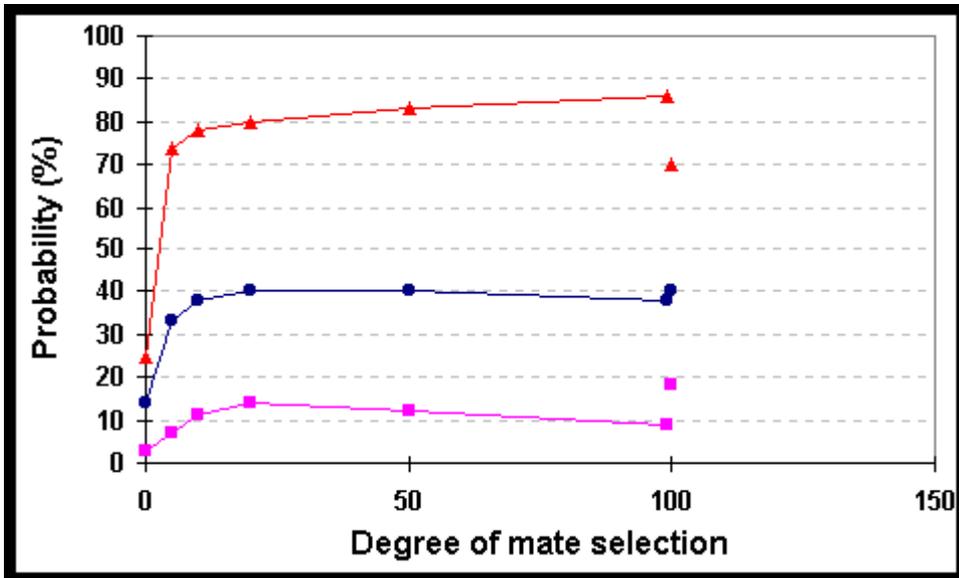
8 The evolution of altruism through parental care

From: Jaffe 2001

 ABSTRACT: What features of biological evolution favor the emergence and maintenance of social behavior? Phylogenetic analysis suggests that parental care of the offspring precedes social cooperation in most taxa. However, present theories focus on genetic relatedness between cooperators as putative evolutionary factors leading to social cooperation. Through computer simulations with Biodynamica I assess the relative importance of reproductive systems (haplodiploidy vs. diploidy), mate selection (assortative mating vs. random mating) and social economics (pay-off matrices of evolutionary games) in the evolutionary dynamics leading to the emergence of social cooperation through the development of behaviors favoring increased parental investment. The results suggest that the most important factors for the emergence and maintenance of social behavior are those based on energetic or efficiency considerations rather than those based on genetic ones. That is, social behavior is most likely to emerge through parental care when the cost for parental investment is much less than the benefits to the offspring, when cooperation improved synergistically the fitness of offspring compared to the corresponding egoistic behavior and when alleles coding for altruistic or social behavior can be rapidly fixed in the population, thanks to mechanisms such as haplo-diploidy and/or assortative mating.

Altruism and social behavior

How social behavior arrived through evolution is still an unsettled aspect of evolutionary theory. A large amount of literature exist on the role of game theoretical mechanisms such as mutualism, of genetic mechanisms such as haplo-diploidy which promote genetic relatedness favoring kin-selection, and economic considerations as putative critical elements in explaining the emergence and maintenance of social behavior, which I will not be able to review here (see review in Crespi and Coe 1997 for example). But none of the studies, to my knowledge, tackles these different aspects together.

Haplo-diploidy or arrhenotoky is known as the reproductive mechanism by which unfertilized eggs produce haploid males whereas fertilized ones produce diploid females. Haplo-diploidy is common among social insects and thus has been proposed as a mechanism which facilitates the evolutionary emergence of social behavior, at least among Hymenoptera, thanks to the fact that it produces an asymmetric genetic relatedness between daughters and sister, increasing genetic relatedness among sisters, and thus favoring altruistic behaviors (Hamilton 1964, West-Eberhard 1975). However, actual field data has proved difficult to explain using this theory (see introduction in Reeve 1993, and review in Crespi and Choe 1997, Jimenez 1998, for example).

Assortative mating favors genetic relatedness among reproductive units. Assortative mating can either be passive, i.e., population viscosity favors reproduction among spatially proximate individuals, or active, i.e., individuals chose their mates based on similar phenotypic traits that reflect similar genes. In both cases it produces more genetically related interbreeding subpopulations. Assortaive mating has been shown to affect the evolutionary dynamics of sexual organisms significantly (Davis 1995, Jaffe 1999a,b, Kondrashov and Kondrashov 1999. Dieckmann and Doebeli 1999) and may thus be a force to take into account when studying the emergence of social behavior.

A game theory approach

Favorable pay-off matrices in game theory may also been invoked as features facilitating the emergence of social behavior (Maynard-Smith 1976, Gomes et al 1999). This argument, presented in thermodynamical terms, states that social behavior has to be more efficient in the exploitation of the habitat, in defense, or in any feature related to fitness, which appropriately measured should be reflected in a more efficient use of energy compared to the equivalent non-social behaviors (see review in Jaffe and

Fonk 1994 for example).

Here I want to answer two questions: 1- Do these features together explain the emergence of social behavior better than each of them alone? 2- Are some of them more relevant than others in favoring the evolutionary dynamics for the fixation of altruistic alleles ? Biodynamica was chosen for this task due to the complex nature of the aim. For modeling purposes, the classical haplo-diploid strategy was decomposed into two components, each specified by the alleles of a gene: the ploidy number of individuals and the inheritance mechanism. Two alternative inheritance mechanisms were modeled: Sexual-Asexual Strategy (SAS), in which males are produced by unfertilized female eggs and females are produced only after sexual reproduction; and Sexual Strategy (SS) were both females and males are produced sexually. SAS males were always haploid, whereas SAS females could be haploid or diploid. Thus, 4 possible reproductive strategies could be simulated: SAS with haploid males and haplo- or diploid females, and SS with haplo- or diploid males and females. For example, a SAS haploid was an organism in which all individuals where haploid and where the males inherited genetic material only from their mothers whereas females inherited a mix of genetic material from both parents. The ploidy number in SS was the same for males and females. These strategies were tested for their survival properties in simulations and for their effect on the emergence of altruistic or cooperative behavior in parental care.

For the study of the evolution of social behavior, a theoretical game was developed. It was based on the eventual evolution of social behavior through development of altruistic cooperative care of the offspring or extended parental care (Alexander 1974, Queller 1994, Crespi and Choe 1997). As all known eusocial or truly social species developed cooperative care of the young prior to reaching full eusociality (Michener 1974, Wilson 1976), the game was developed to compare the evolutionary dynamics of 5 parental investment strategies. Brood care could be provided by both sexes or by only one of them (according to alleles of the corresponding gene). The strategies for brood care, coded by the corresponding alleles, were designed as idealized strategies, whose mixes cover most of the behaviors based on parental investment, known to exist in nature. These were:

No care: Parents did not invest in offspring. (This probably represents an extreme situation as even in species with no parental care, mothers invest in their offspring prior to laying eggs or giving birth)

Communal care: Parents invested equally in all offspring of the population, irrespective of genetic relationships. This strategy models extreme alloparental care. Alloparental care is fairly common (for example among coleoptera: Scolitydae and Curculionidae, and communal wasps).

Open care: Parents invested half their available energy in their own offspring and half in all other offspring. This represents a mix of alloparental care with selfish care (for example social primates, ungulates and wasps).

Opportunistic care: Parents invested only in their own offspring, but their offspring received half of the care from open or communal parents. This strategy appears to be used by species breeding in communal breeding sites (for example among birds, sea lions, elephant seals).

Closed care: Parents invested only in their own offspring and their offspring could not receive care from any other adult. (for example digger wasps, scorpions, dung beetles)

Each organism possessed a gene defining the "degree of parental investment" whose allele coded for the amount of energy spend in caring for the brood . Each parent investing in offspring suffered a cost to is fitness which was directly proportional to the "degree of parental investment" multiplied by the cost/benefit ratio of investment. The cost/benefit ratio of parental investment was constant for each simulation and determined the ratio between the cost in fitness to the parent making the investment and the benefit in increased fitness to the offspring receiving the care. The increased fitness of offspring receiving parental care was directly proportional to the "degree of parental investment" only.

A constant simulating the benefit of sociality ("social synergy") determined the amount of synergy achieved by social behavior. This "social synergy" increased the increase in fitness the offspring received from caring adults if the care was communal, and half that amount if the care was open, but not if the care was opportunistic or closed. That is, I assumed that parental investment was more efficient with cooperation. This assumption can be illustrated taking as an example two extreme situation of brood care. More time dedicated to caring the brood will reduce the odds of parasites or predators killing the offspring. If one parent invest 50% of its time to care for its brood, it will leave the brood unattended 50% of the time, and the odds of loosing the brood (p = 50 * k, where k is a proportionality constant). If two parents attend their brood communally, each one investing 50% of their time in brood care, the brood may be protected 100% of the time, reducing the odds of loosing their brood to zero with the same cost to parents. In this example the value for "social synergy" is $50*k / 0 = $ infinite for k > 0. A "social synergy" value of 1 indicates no advantage of communal care over egoistic parental investment.

The relative importance of genetic and energetic considerations

In order to assess the effect of diploidy and haplo-diploidy on the likelihood of altruistic genes being fixed in a population, "gene selection" experiments were performed. The simulations compared the ability of each of 5 alleles, coding for different parental care strategies, in displacing the others. These experiments were performed with haplo-diploid and pure diploid organisms using either assortative mating or random mating.

Simulations of extreme situations gave the expected results. That is, for values of cost/benefit ratios of parental investment greater than 1 (i.e. the cost to the parent is much greater than the benefit to the offspring), alleles coding for no parental investment displaced all others. Values of social synergy less than 1 made that alleles for egoistic (opportunistic and closed) parental investment displaced all others.

Simulations using values of cost/benefit less than 1 and of social synergy greater than 1, gave interesting results (Fig 8-1). Each combination of reproductive system and mate selection strategy produced a different evolutionary dynamics of the allelic frequency for the gene coding for parental care. In all cases, the extreme strategies, i.e. communal care and closed care, displaced the intermediate strategies for parental investment. The allele coding for closed care rapidly increased its frequency in populations using random mating in both halpo-diploid and diploids, but in the long term (200 time steps) alleles coding for communal care became the most frequent, except in diploids using random mating. Strategies using assortative mating fixed alleles coding for communal care faster than those using random mating. This dynamic was influenced by the cost-benefit ratio of parental investment and by the amount of synergy social behavior could achieve (Fig 8-2 and 3). That is, at high cost/benefit ratios, alleles coding for closed care were more likely to be fixed in the population, whereas high values of social synergy favored communal care. Assortative mating favored the invasion of alleles for communal care at higher cost/benefit and lower social synergy values than random mating.

Also haplo-diploidy favored the invasion of communal alleles at higher parenting costs and lower social synergy values than pure diploids, but to a smaller degree than assortative mating. Thus, populations of haplo-diploid organisms using assortative mating could fix alleles coding for communal care at about twice the cost/benefit ratio for parental investment and about half the social synergy value than diploid organisms mating at random. Simulations showed that Haplo-diploidy drives evolution with a faster dynamics than pure diploidy, confirming that the simulations are in agreement with the existing literature (Mable and Otto 1998). This special ability for fast adaptation of a complex assemblage of genes is a general feature of haplodiploidy, not restricted to the evolution of social behaviors, and is due to the increased exposure of alleles in haploid males. This mechanism may explain why haplo-diploidization technologies improve crop selection (Goldringer et al 1996), and why haplo-diploids populations establish faster resistance alleles than diploid-diploid ones (Caprio and Hay 1995). The fast diffusion of traits linked to haplo-diploidy is due to advantages of haplo-diploid female genes for generating uniparental males (Bull 1983), again showing that the results of the simulations are in agreement with existent data and theory.

Assortative mating increases genetic relatedness in the population favoring kin-selection. Assortative mating is also a strong evolutionary driving force favoring the emergence of behaviors with long-term fitness increases, through mechanisms different to kin-selection (Hamilton 1964). The present results show that assortative mating favors the emergence of altruistic behavior, but the simulations do not allow to pinpoint the importance of close genetic relatedness in this process. As genetic relatedness is not likely to explain the evolution of social behavior, at least among hymenoptera (Queller 1988, Gadadkar 1990, 1991), we might assume that the positive effect of assortative mating in accelerating the fixation of altruistic alleles, observed in the simulations, is due to rather more general dynamic properties of assortative mating analyzed in detail above.

The results suggest that economic considerations (the cost-benefit ratio of parental investment, the amount of synergy achieved by social behavior) are the most important factors explaining the emergence and maintenance of social behavior, followed with decreasing importance by assortative mating, and haplo-diploidy. Interestingly, the model revealed the obvious but neglected fact that social behaviors which provide no social synergy in increasing the fitness of the offspring are unlikely to emerge in biological evolution. Social behavior should emerge in evolution when cooperation or altruism improves synergistically the fitness of offspring compared to the corresponding egoistic behavior, and when alleles coding for social behavior can be rapidly fixed in the population, by strategies such as haplo-diploidy, assortative mating, and probably others (Simon 1990 for example).

The results of these simulations suggest further that social behavior is unlikely to emerge when the cost to the parents providing investment is much higher than the benefits received by the offspring, favoring egoistic varieties of parental investment instead. Generalizing this idea, we might postulate that social behavior in general emerges and is maintained when the economics of sociality provides larger fitness gains compared to that of individual solutions. Experimental results seem to support these predictions. Among examples of interactions between Lycaenid (Lepidopera : Lycaenidae) larvae and ants, cooperation is much more common than exploitation (Osborn and Jaffe 1977). No kin-selection argument can be given for explaining social cooperation between these non related taxa, suggesting that rather economic considerations may explain the frequency of those cooperation's. Other evidence for the importance of social economics are measurements comparing the energetic efficiency between isolated individuals and social aggregations or between small and large societies, which are going to be discussed in the next chapter. In conclusion, the simulations, in congruence with the scant experimental evidence available, suggest that economic considerations rather than genetic ones are critical in explaining the emergence and maintenance of sociality.

Experimental parameters used

The characteristics of the organisms subjected to selection were as follows: Maximum life span = 10 time steps, clutch size = 3 offspring / female, minimum reproductive age for males and females = 1 time step, number of males screened by females searching for potential mates = 20 for assortative mating and the first male encountered in random mating, mutation rate = average of one mutation every 25 genes in every newborn, both sexes provided equally for parental investment. Genes with various alleles coding for different phenotypic expressions were inheritance mechanism (SAS or SS) where all organisms were diploid except males in SAS, and strategy and degree for parental investment.

The population endured the five-step transformation, at each time step, representing one reproductive cycle. Generations overlapped as each female reproduced independently. Thus, one time step corresponded, under the life history traits simulated here, approximately to 0.2 generations (i.e. 5 time steps corresponded in average to one generation). The exact genetic composition of the population was plotted at each time step, with the actual number of surviving individuals. Thus I could assess when and if the population reached a stable population size, and at the same time monitor the genetic composition of the population at each time step.

Simulation aimed at assessing the effect of selection on the allelic distribution of a given gene in a

single interbreeding population. For example, alleles coding for different parental investment strategies were assigned at random but uniformly among the first individuals. The simulation was started and the frequency in the population of the various alleles was monitored for each time step. In experiments designed to assess the evolutionary stability of certain strategies, the initial population had individuals, all with the same allele for that strategy. After 20 time steps, mutant alleles coding for different strategies were allowed to appear, and the frequency of the two alleles was monitored for at least a further 400 time steps or until the mutant allele displaced the other.

See Demo08, Demo15 and Dem016 of Biodynamica

Figure 8-1: Temporal dynamic of the relative frequency of alleles (frequency) of the gene coding for strategy of parental investment for haplo-diploids with random mating (HD-R), haplo-diploids with assortative mating (HD-A), Diploids with random mating (DP-R) and diploids with assortative mating (DP-A). Values for social synergy = 3, degree of investment = 50 % of fitness, cost/benefit ratio = 0.3. Each data point is the average of 200 simulations.
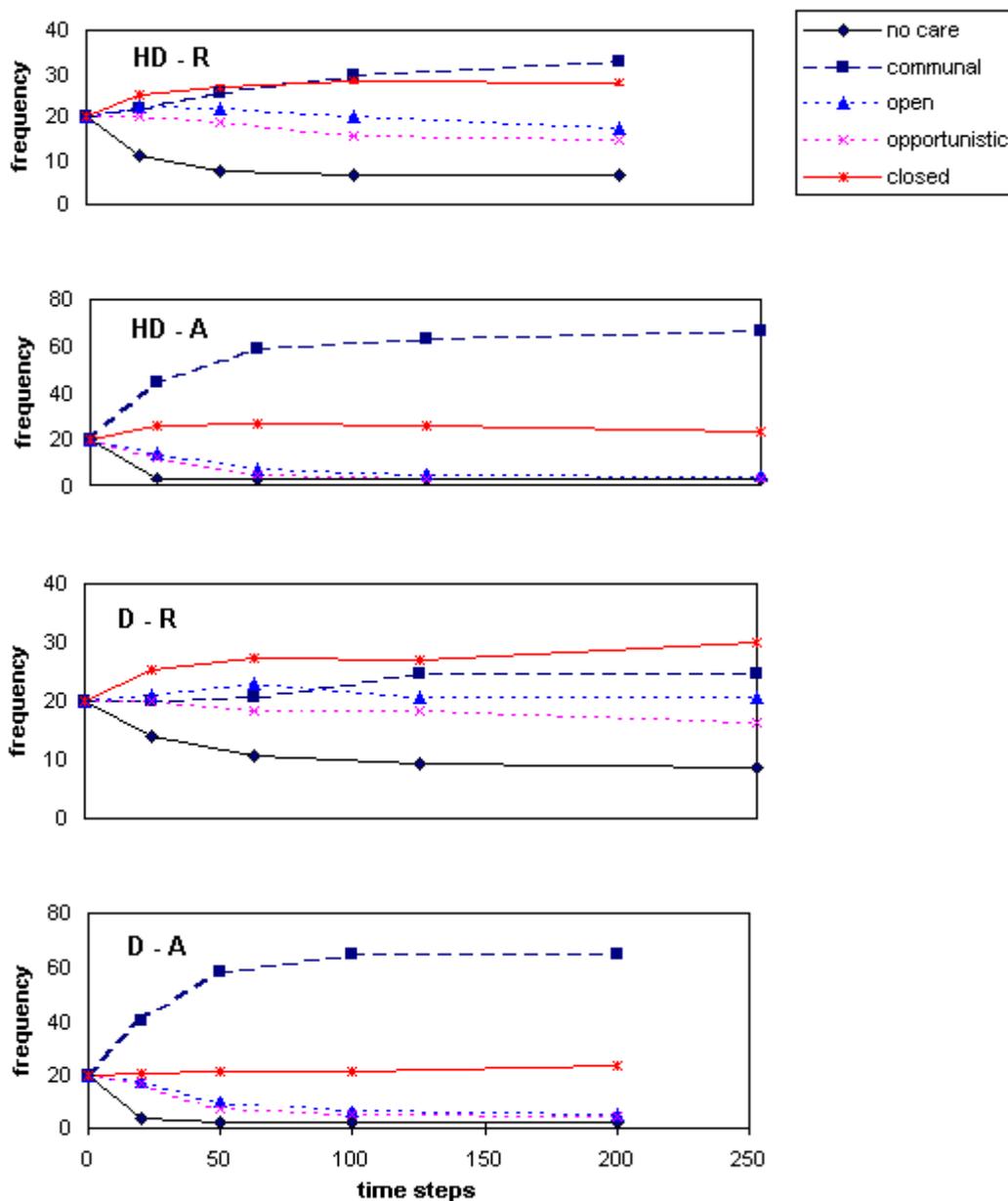
Figure 8-2: Effect of the cost/benefit ratio on the probability of finding an allele coding for communal investment in an individual of the virtual population after 200 time steps. Values for social synergy = 4. Else as in Fig. 8-1
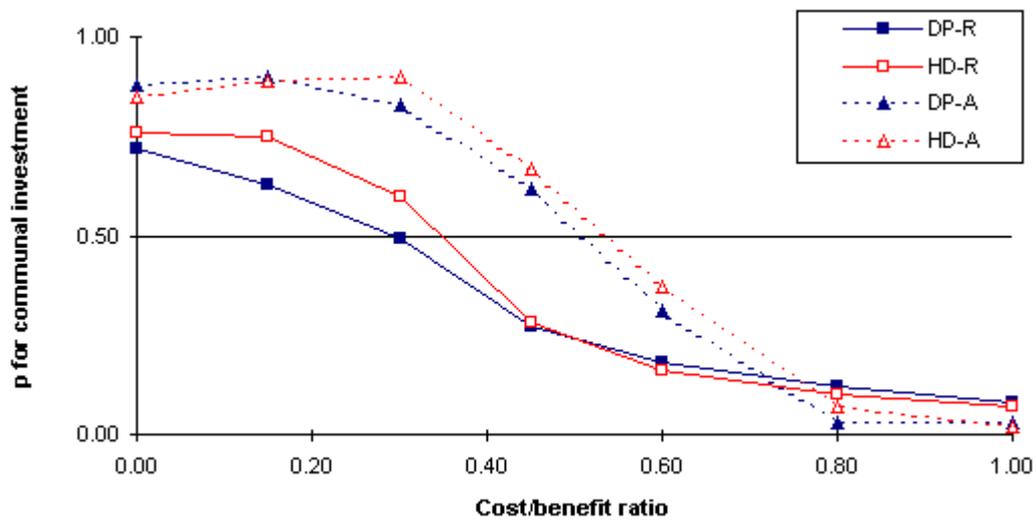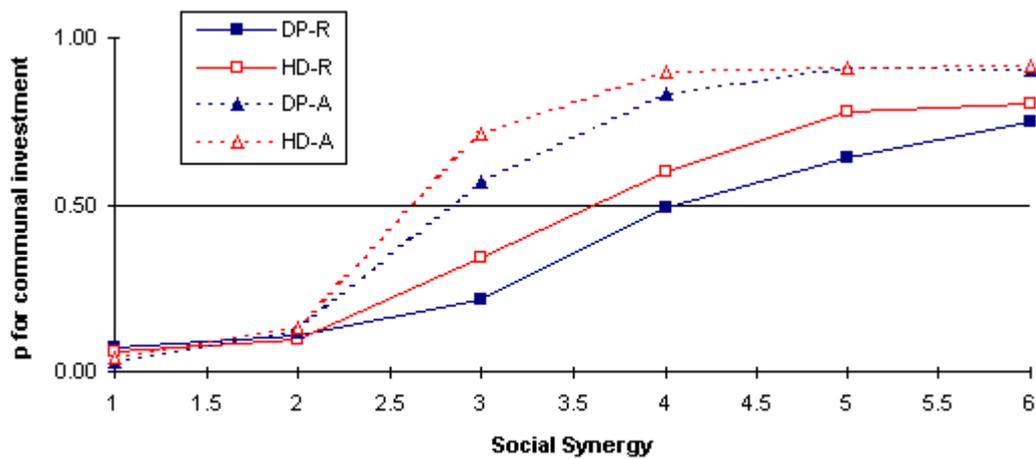

Figure 8-3: Effect of the social synergy on the probability of finding an allele coding for communal investment in an individual of the virtual population after 200 time steps. Values for cost/benefit ratio = 0.3. Else as in Fig. 8-2

9 Biological Thermodynamics and Evolution of Societies

ABSTRACT: According to thermodynamic considerations, complex systems may be comprehensively described in terms of their energy balance. Data of energy consumption per individual of societies of ants, termites and humans showed that energy consumption is a reliable estimator for biological relevant indices. Experimental data on energy consumption in these animals suggests that the relationship between energy consumption and social complexity is non-linear. Societies smaller than a certain critical size have energy requirements that are larger than the sum of the average energy requirements of isolated individuals. Societies larger than this critical size show social optimization mechanisms, by which larger conglomerates or societies are more efficient in using energy per individual than smaller ones. Thus, social complexity is related to energy consumption in a discontinuous manner and is bound by both, negentropy content of a society and social optimization mechanisms.

Energy as the most important thermodynamic variable in biology

The thermodynamic approach allows the study external expressions of a system, even if the underlying dynamic mechanisms are not tackled directly. Thus, the temperature of a system may have enormous predictive value, even if the kinetic energy of each of the components is not known. Measurements of energy allow us also the estimate the entropy (or negentropy) of a given system, which in turn allows us to infer about the degree of order or disorder reining in it. This approach, common to chemistry and physiology, has rarely been deployed in evolutionary biology (but see Brooks and Wiley, 1976). Yet, the few biological examples showing the possible relevance of such an approach in the study of evolution, seem to me very significant. For example, the use of the negentropy concepts allowed improving our understanding of the evolution of chemical recruitment in ants (Jaffe 1984). In this study, the calculus of the amount of information communicated via chemical means allowed to discern the phylogeny of chemical communication systems in ants.

Regarding the properties of social systems, from a thermodynamic perspective, the following questions seem relevant to the understanding of the evolution of societies:

i. Is the increase in social complexity, measured as the number of individuals living in a given society, correlated to an increase in efficiency of energy use by the society (optimization principle), or
ii. is the increase in social complexity related to an increase in energy consumption per unit of biomass (thermodynamic prediction)?, as more complex systems require more energy for their maintenance

The answer to these questions is relevant for the choice of variables, to be used in sociometrics and econometrics, for the assessment of important dynamic features of societies.

Biodynamica showed that energy efficiency may be an important element in directing or aiding evolution towards complex societies (see previous chapter). Other optimization theories derived from biological theory supposes that evolution tends to create ever better-adapted organisms (or societies). In this context, if sociality is considered to be a higher form of expression of life and thus a sophisticated product of evolution, societies should optimize the use of energy, as can be extrapolated from various optimization principles proposed for biological systems, such as optimization of average fitness (Dobzhansky 1951), maximal efficiency in resource utilization (Macarthur 1962), minimal metabolized energy per unit biomass (Hannon 1979), maximum energetic power (Torres 1991), minimal rate of entropy dissipation (Nicolis and Prigogine 1977). On the other hand, irreversible thermodynamics predicts that as social systems represent higher level of complexity compared to non-social systems, they are further away from a stationary state, and consequently require more energy for their maintenance (Jaffe 1984, Jaffe and Hebling-Beraldo 1993, Lamprecht and Zotin 1978).

Formulation of the problem

Essentially, three hypotheses should be considered when investigating the possible relationship between the degree of social organization of a society and its energy consumption:

a) Optimization hypothesis: Social life is energetically more efficient, resulting in a reduction of energy expenditure per biomass unit. Given that solitary life precedes social life, social organization should result in optimization of energy use, as societies adapt to environmental constraints. More evolved or adapted societies should thus consume less energy per capita than more primitive ones.

b) Thermodynamic hypothesis: Social life implies an increment in energy costs per individual. In this sense, the social unit would be more than the sum of its parts. Such an outcome would be a prediction of irreversible thermodynamics theory applied to dissipative structures, i. e. living systems. The society with its members and their interactions forms a complex system that maintains a certain organization, thus requiring external energy proportional to the degree of "orderliness" or negentropy of the society. Thus, more evolved societies should require more energy per individual.

c) Neutral hypothesis: Social life has no effect on the energy consumed by members of the society; the energy intake of the whole society equals the sum of the consumption of each individual, which is similar to that of an isolated individual. This is normally assumed among biologists for estimates of the energy expenditure (Brian 1978, Godwin 1982, for example)

The experimental evidence

These hypotheses have been tested experimentally in ant societies (Jaffe and Fonk 1994, Fonck and Jaffe 1996), in termite colonies (Muradian 1997) and in human societies (Cabrera and Jaffe 1998). The main results are shown in Figures 8-1 to3. Figure 8-1 shows the energy requirements per unit of biomass of ant aggregates. Individual ants vary greatly in their energy use (group of points at the left of Figure 8-1). Once grouped, energy requirements seem to converge to an average value (points around the horizontally fitted line in the figure). After a certain critical number of ants aggregates, a bifurcation point is reached. Aggregates at or above the bifurcation point consume significantly more energy (points around the fitted line on the right of the figure), probably due to the fact that the aggregate starts functioning as a society, where ant start engaging in nest-building activities, etc. Increasing the number of ants after that critical point reduces exponentially the energy requirements per unit mass. In Figures 8-2 and 3 whole termite colonies or stabilized human aggregates were studied. Thus, the system is already stabilized and is equivalent to the last phase of the ant aggregates of Figure 8-1.

Based on these results we may conclude that there is a complex non-linear relationship between colony size and energy consumption per unit mass in these social insects. Results showed that after a certain critical colony size, the colonies energy consumption is larger than the sum of the average energy requirement of the individual workers, and near maximal colony sizes, the energy consumption of the colony per unit mass tends exponentially to that of the average individual worker. It seems therefore that social complexity is related to energy consumption in a discontinuous manner and is bound by both, the negentropy content of a society (the thermodynamic hypothesis) and social optimization mechanisms (the biological hypothesis). The thermodynamic hypothesis seems to apply for the formation of societies or when comparing social systems with non-social ones, whereas the biological hypothesis applies to comparisons of societies of different degrees of development or size.

In the case of ant societies, further experiments showed that the critical value after which social optimization processes start to work differs for different species and is probably related to the blend of

energy efficiency of the different worker castes forming the society (Jaffe and Hebling-Beraldo 1993).

Social thermodynamics

Based on the experimental evidence of three totally different social organisms we may reach the following conclusions:

1- The generally assumed neutral hypothesis is not valid.

2- The energetic cost of assemblages of organisms per unit of biomass is a function which is unknown below a minimum threshold size of villages, above which it decreases exponentially with increasing size of the system.

3- Normally functioning societies optimize the average energy consumption per individual as the society increases in size, where larger societies are more efficient in optimizing energy use.

Although alternative interpretations of the data and different statistical analyses could produce different conclusions, this work suggests that a general phenomenon, applicable to human and insect societies, may exist, which affects the energy consumption of the system, as energy balances allow to estimate the energetic cost of sociality in a given system. This work demonstrates the heuristic value of applications of irreversible thermodynamics to biological and social systems for the detection of new bio-social phenomena. The approach though does not allow pinpointing the casual relationships explaining the variance in energy consumption. They only serve to pinpoint general constraints of complex systems and should be complemented with detailed sociological and economic analyses in order to get a better understanding of complex societies. We propose that energy consumption as an econometric index may reflect socio-economic development better than alternative indices.

Figure 8-1. Relationship between oxygen consumption and size of ant aggregates in three different ant species. (From Jaffe and Fonck 1985 and Fonck and Jaffe 1996.)
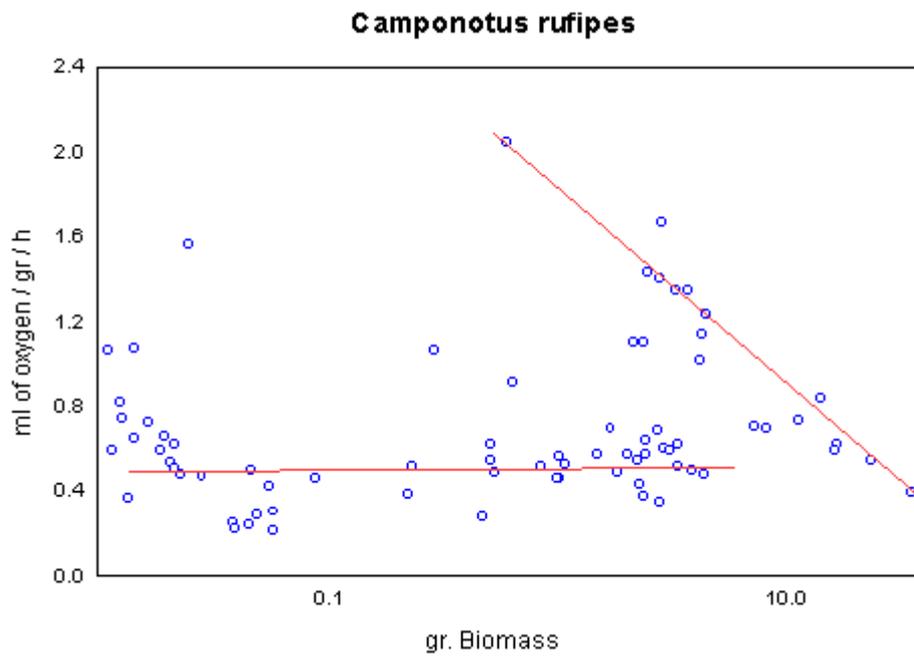
Camponotus rufipes
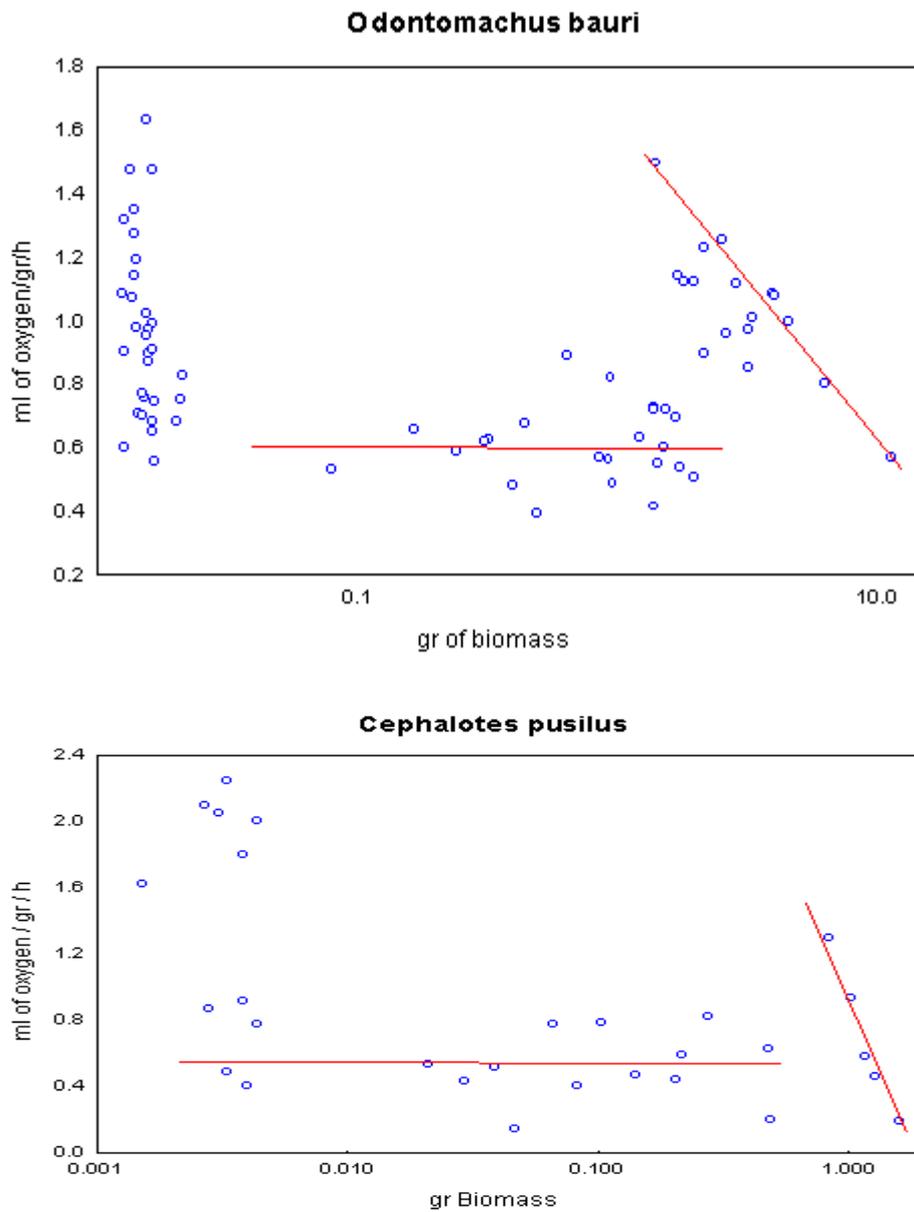
Figure 8-2: Relationship between CO2 production and colony size in the termite *Nasutitermes ephratae*. (Muradian, Issa and Jaffe 1999)
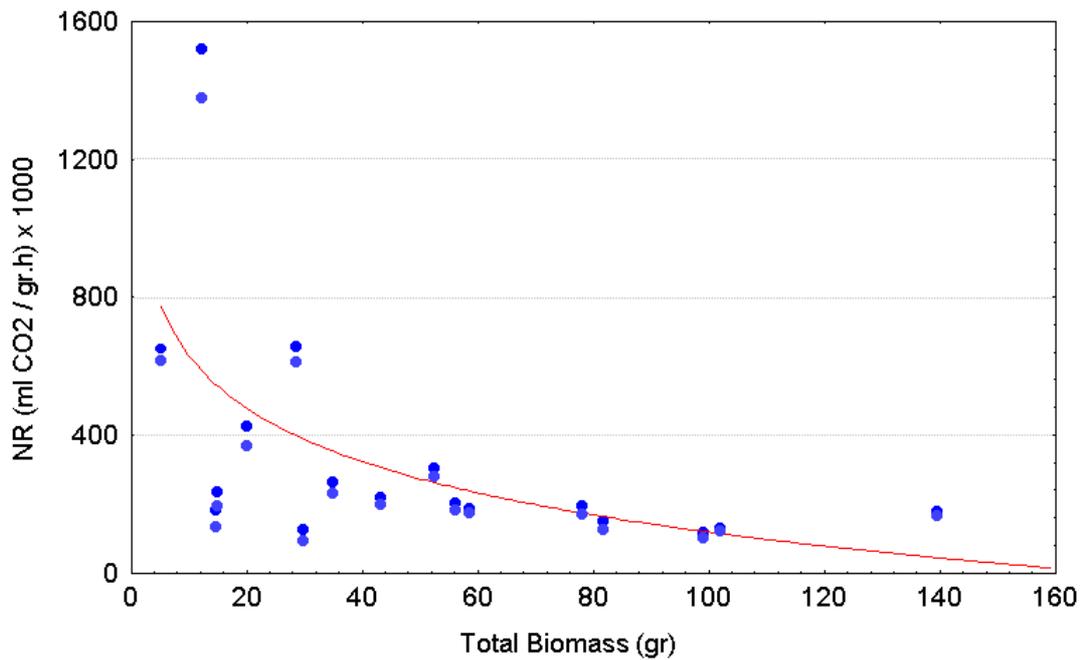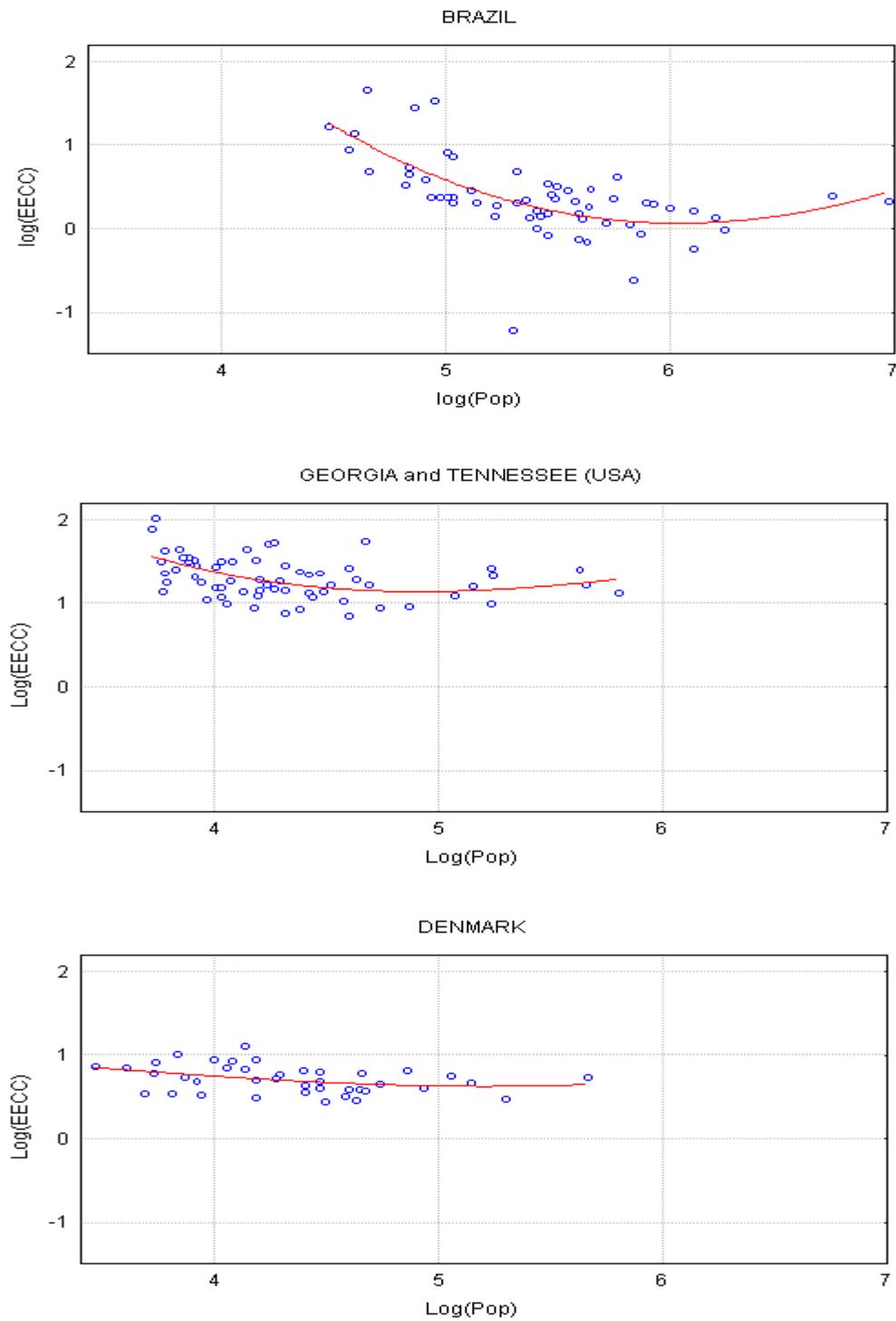
Figure 8-3: Relationship between electricity consumption and size of urban human settlements in three different countries (Cabrera and Jaffe 1998)

9 Perspectives for a probabilistic quantitative evolutionary science

The study of complex dynamic problems often cannot be achieved by using the classical techniques of the scientific method. That is, analytical mathematical conceptualization of the problems and experimental designs to conform or reject predictions, based on these conceptualizations, is not always possible in astronomy, geology, meteorology, evolutionary biology, economics nor human history. These are all sciences in which the classical design of experiments, where all variables are controlled, is impossible. The study of naturally occurring experiments and the use of the comparative method, of diverse multivariate analyses and of computer simulations seem therefore to be the tools for these sciences.

Evolutionary theory specifically and all historical sciences in general, in order to become more scientific as disciplines, have to make themselves eventually more quantitative and predictive. One such approach, as I suggest with this book for evolutionary theory, includes the calculus of probabilities or feasibility of certain processes, and based on those qualitative and quantitative estimates, the theory can be tested for accuracy with field data. I showed that for the support and development of evolutionary theory, field data might be obtained through thermodynamic studies of living systems or through the assessment of the frequency of occurrence of a given strategy among the various taxa. Certainly other methods should be developed. I view the future of evolutionary biology as a synthesis between sciences such as Artificial Life, computer simulations of real systems, phylogenetic analysis, biological thermodynamics, evolutionary physiology, complex system analysis, economic sciences, sociobiology and psychophysiology, among others.

What may we conclude from the results of the computer simulations?

The limitations of our mind to follow the dynamics of a multitude of interdependent variables that act simultaneously on the object of study, can be overcome with the aid of computers. Complex problems in evolutionary biology and other historic sciences can be analyzed with computer simulations and insight can be gained about the relevant dynamic interactions involved. Ever more complex models may provide ever more relevant insights. I showed how computer simulations might aid our understanding of the emergence and maintenance of sex and of altruism. Specifically, the working of assortative mating in evolutionary dynamics showed that genes might penetrate and dominate populations without conferring a measurable fitness to the organisms possessing them. The simulations also highlighted the irreversible and stochastic nature of evolutionary dynamics. If several features, such as sex and altruism, are incorporated into the genetic repertoire of species through specific dynamic advantages, it is difficult to ascribe to the process of survival of the fittest all the genetic features we observe in extant and extinct organisms. The term luck seems more appropriate to describe this process as it forces us to look for some more complex dynamic processes to describe and understand biological evolution. If we agree that luck, or for that case, any stochastic phenomena, affect the outcome of evolution, a statistical approach to the study of evolution seems the only rational way forward. Thus, future biologist will certainly be more willing to speak about the probabilities of certain outcomes to occur in evolution, and thus treat biological evolution as a quantitative statistical science. This may increase enormously the predictability of evolutionary biology, eventually leading to the emergence of ecological engineering: i.e. a rational dynamic manipulation of complex biological systems.

Simulations may be very powerful in helping our understanding of the evolution of societies and of economic phenomena in general. Some of the questions which should be tackled with future models include: Why is specialization a basic feature of all known evolutionary process leading to sophisticated social systems? Why are individuals in complex societies simpler and more specialized in the task they perform than non-social organisms? Other problems of evolutionary biology such as co-evolution, the dynamics of birth and dead, life history traits, speciation, macroevolutionary dynamics, among many others, may also be amenable to similar treatments. The future certainly offers an important role for

computer simulations as theoretical and experimental tools for the study of complex systems in biology.

New experimental studies should also help in improving the modeling of biological evolution. Quantitative information about the number of species possessing certain strategies may help in detecting attractors in evolutionary dynamics. For example, we could produce quantitative data on the number of sexual, asexual and facultatively sexual species in each taxon, which would help our search to find conditions when these strategies may stabilize in evolution. Information about the number of species having no social behavior, having advanced social behavior, and those having intermediate degrees of sociality, may be indicative of which social traits develop fast, tending to evolutionary equilibrium, and which traits are less affected by dynamic constraints, allowing species to maintain those intermediate traits.

Towards a quantitative evolutionary biology

In order to improve the predictive power of evolutionary theory, we need to produce testable predictions that are more quantitative in their character. Such improvement will require the building of ever more complex evolutionary models, made possible by the ever greater power of computers. For example, models like Biodynamica may predict the probability of occurrence of certain mate selection strategies or sexual reproductive system under certain environmental conditions, for example, and paleontological and ecological data may be used to check on the accuracy of the frequency of occurrence predicted for the specific solutions

At the moment though, I do not expect that theoretical models will be sophisticated enough to achieve reliable quantitative predictions. Experimental evolutionary biology with virtual and real organisms seems to be more feasible in the short term. That is, experimenting with evolution in artificial worlds, i.e. Artificial Life, may provide qualitative predictions that may be tested, confirmed or rejected with ecological and paleontological data, and may prove to be a more feasible approach. Experiments with fast breeding real organisms, will certainly also help us in gaining insight about the processes guiding biological evolution, helping our culture to acquire a better understanding of the problems awaiting the future of humankind.

Towards a synthesis of historical sciences

The study of human history, economic phenomena, political changes, technological diffusion, marketing and sociological processes, share with evolutionary theory their methodological shortcomings. All the sciences involved in studying complex dynamic multivariable phenomena may eventually profit from a greater exchange of techniques and methods used in their scientific studies. Such an increase of interdisciplinary activity should be purposely planned and encouraged as the challenge to tackle complex dynamic processes is probably above the possibilities of any single discipline. In this respect, I have great fait in computer simulations. I hope that this book may contribute to advance the endeavor of eventually unifying biological evolutionary theory with modern human sciences. Various such attempts, using completely different methods (see for example, Tudge 1996, Diamond 1999), may hint that human intelligence may be ripe for this new synthesis.

Any new approach to complex system sciences, I think, will incorporate conceptual elements proposed by past thinkers. Among them I would like to steer the reader's attention to Abu Jafar Muhammad's algorithms, to Alexander Bodanov's Tektology, to Ludwig von Bertalanffy's General System Theory, to Maturana and Varel's cognitive autopoiesis, and to Kurt Gödel's insight into the cognitive limitations of any system. By mixing these ideas and extracting a synthesis, I visualize a future system science in which theories will become tools (computer models or algorithms) for cognitive heuristic explorations, which by their own will be incapable (thanks to Gödels theorem) to advance their own systemic improvement (autopoiesis), requiring a constant input from external sources, such as results from experimental and other natural sciences, in order to advance, improve and increase their predictability. This approach is basically the same as the scientific method used in traditional sciences, it

is only more complex in that the cognitive actions are carried out in a systemic hierarchy, where theories are ever more complex models, where experiments are ever more sophisticated comparisons of natural phenomena and where more and more disciplines will participate in this scientific endeavor. The final aim of this effort is to achieve a continuous increase in the explanatory power and the predictability of models and theories of complex systems, and thus to increase our power over our future, augmenting our luck.

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